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EDITORIAL

MODERN TRENDS IN TREATMENT OF FRACTURES*

THE histories of all branches of medicine are characterized by eras of activity and advance followed by periods of relative quietude. During the first part of the active period there is abundant development and enthusiastic broadcasting of new ideas, new methods and new instruments. This is accompanied by heated discussions between proponents of the new and adherents of the old. Next comes a period characterized by judicious examination, weeding out unworthy methods and consolidation of the gains made. During this process certain fundamental principles may be thought to rest on faulty premises. The principles which are discarded are replaced by new fundamentals which may prevail only until the next period of activity. Then the structure must be rebuilt in the light of advanced knowledge or new discovery, occasionally with a return of the old principles. The length of the periods of inactivity may depend upon many factors. They may be ended by the advent of a dynamic genius such as Harvey Cushing was to neurological surgery, or by the collective efforts of many as in abdominal surgery. Discovery in a collateral chemical or physical science, as in the field of radiant energy, may be the necessary spark. Occasionally better advantage is suddenly taken of known physiological facts. This is illustrated by the recent appreciation of pressure relationships within the thorax and their control by differential pressure anesthesia. Again the invention of some mechanical device may produce great changes. Witness the effect of the cystoscope in urology, the silver clip and the endotherm in surgery of the brain, and the general result of the introduction of the clinical thermometer and the stethoscope. Even the Murphy button, all but forgotten within thirty years, was a device which did more to

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stimulate progress in intestinal surgery than, perhaps, any other single factor.

Surgical therapy in general was held up until the latter part of the Nineteenth Century in order to complete its necessary triad of fundamental principles, hemostasis, anesthesia and asepsis. Enormous strides have been made since that time and not the least part of the advance has been due to mechanical aids placed at the disposal of the surgeon. Well into the beginning of this century the operating theatre was often in the home, sometimes a farm house, with the patient resting on a kitchen table or on a window shutter supported by two chairs. If at night, the anesthetic, which was frequently administered by one of the family, was chloroform because of the open flame of coal oil lamps. The few clumsy instruments were boiled on the kitchen stove and the field was draped with sheets sterilized in bichloride solution. My earliest surgical recollections are of watching and later assisting my father under such circumstances. Granting that the surgery done in such surroundings was good surgery what surgeon of today would willingly return to them? In contrast, visualize the modern, well lighted operating room with its sturdy, flexible table, efficient anesthesia apparatus and large array of instruments, each designed to perform effectively a given function. Some of these special appliances, particularly the various endoscopic operating instruments, have reached a high degree of mechanical perfection. All these devices are backed by the modern sterilizing, *x*-ray and laboratory facilities of the well equipped hospital. It is fair to say that for the past forty-five years surgery owes quite as much to improvements in its mechanical armamentarium as it does to increased knowledge of fundamentals.

The treatment of fractures has been almost alone in lagging behind the general progress of surgery. Little real advance has been made since Hippocrates.[1] The Father of Medicine minutely described methods of reduction and fixation which are in general use today. His directions and positions for traction and counter-traction are paraphrased by most modern texts. He used a fracture table which was similar in essentials and design to the Hawley table. The basic principle of the Thomas splint, its intrinsic traction with counter-traction applied at the proximal end of the extremity by a padded ring, was employed by him. He used casts made of bandage inpregnated with wax instead of plaster of Paris. He was guided by two fundamentals, first, "All the bones get consolidated more slowly, if not laid properly, and if not kept steady in the same position"; and second, "It should be kept in mind that exercise strengthens and inactivity wastes." These two principles stand firm as the true fundamentals of the treatment of fractures, despite all the tinkering of the past twenty-four hundred years.

What improvements were made on the methods of ancient Greece at the time when other branches of surgery were coming into their own? Following the advent of asepsis and the *x*-ray, numerous techniques for open reduction and internal fixation immediately blossomed. End results were sometimes improved but still far from ideal. Except for the patella, the olecranon and possibly the neck of the femur, have the results of open reduction shown that it is an advance that will stand the test of time? Are not the Lane plate, the Parham-Martin band and other appliances for internal fixation following the Murphy button onto the shelves reserved for surgical curiosities? Traction-suspension methods with such accessories as the Thomas splint also became popular and have played a predominant part in treatment. Their evolutionary importance cannot be overestimated, but the methods have serious defects. The apparatus used is crude, cumbersome and mechanically inefficient; neither reduction nor fixation is accurate or positive, requiring constant readjustment and hospitalization is pro-

[1] The Genuine Works of Hippocrates, Francis Adams, William Wood & Co., 11: 7–179.

longed as the patient is neither ambulatory nor portable. Until recently the *x*-ray, local anesthesia and skeletal traction were almost the only real improvements that modern science had made over the technique of the Greeks.

Meanwhile, the fundamentals of Hippocrates had been all but forgotten. Not only the fracture but the entire limb and often the patient were immobilized in cumbersome splints, casts, or traction-suspension appliances, frequently without true fixation of the bones. The fact that "exercise strengthens and inactivity wastes" was completely ignored. Or, in an effort to provide pseudo-exercise and artificial stimulation, resort was made meddlesome manipulations, frequently causing pain and disturbing the bones which were "not laid properly" and "not kept steady in the same position."

Any engineer would be astounded at the crudeness and mechanical inefficiency of the usual appliances used for the reduction and fixation of fractures. Stripped to its essentials, the reduction and fixation of a fracture (satisfying the first of the fundamentals of Hippocrates) is a problem in mechanical engineering involving the control of known forces transmitted to the fragments through the muscles attached to them. As usually executed it lacks most of the precision of other mechanical arts and certainly does not compare favorably with the accurate technique employed by the expert machinist, cabinet maker or dentist. Comparison of the everyday mechanical appliances used in the home, office or shop to the crude apparatus used to treat fractures in our best hospitals supports this. No machinist would be satisfied with equipment so cumbersome and inefficient as the usual Balkan frame with its dependant maze of cord, pulleys, weights, adhesive, splints and bandages, all for a purpose which, from its mechanical essentials, should not be much more complicated than many of the more simple mechanical processes.

The work of Boehler,[2] following his experience in the war and in the operation of a fracture clinic where results are evaluated on accurate mathematical and even financial estimates of final function, has opened a new era in the treatment of fractures. Boehler has returned to the Hippocratic fundamentals, expressed by him as follows:

1. In every fracture the displaced bone-ends must be correctly replaced.
2. The corrected fragments of the fracture must be retained in correct position until bony union has occurred.
3. Whilst the necessary fixation of the replaced fragments is maintained, many or all of the joints of the injured limb and of the whole body are given a full range of movement as far as this can be done without causing pain.

In applying these principles Boehler and others working along the same lines have attempted to devise technical methods and mechanical appliances which will approach in effectiveness the methods and devices used in other mechanical arts. These attempts have resulted within the past ten years in a multiplicity of techniques and a vast array of instruments and apparatus, some crude and complicated, others simple and efficient, but none perhaps completely satisfactory. Methods are being devised and tested, to be discarded or adopted, with astonishing rapidity and the intelligent observer who wishes to keep abreast of the times is bewildered by a mass of contradictory evidence. Such a condition is natural and inevitable at this stage of development. A similar state of affairs exists in the fields of pulmonary and prostatic surgery. Next will come the time of comparison, discussion and consolidation with eventual attainment of the immediate objective. *This objective is the application of an adequate external scaffold which will completely and functionally replace the broken bony framework until repair has*

[2] The Treatment of Fractures, Lorenz Boehler, 4th English Edition translated by Ernest W. Hey Groves. Baltimore, William Wood & Co., 1935.

been completed. It will provide accurate reduction and firm fixation of fragments, allowing at the same time complete function of the entire locomotor apparatus including bones, muscles, joints, vessels, nerves and skin. The details of the method ultimately adopted will probably be a combination of the best points of the numerous techniques advocated, but the exact formula cannot be predicted. It is safe to say, however, that the final method will adhere to the fundamentals of Hippocrates and will employ means which take advantage of the efficiency of modern mechanical processes.

R. A. Griswold.

A CORRECTION

In the February issue of the Journal on page 209, in an article by Lewis G. Cole, the following quotation appears:

"that all organic lesions involving the pylorus within one inch or so of the pyloric valve should be considered malignant until proved otherwise."

This is not a true quotation and appearing as it does it is misleading. As you will see from the enclosed abstract of my paper published in the Journal of the American Medical Association, September, 1932, the true quotation should read:

"any chronic, indurated ulcerating lesion occurring in the pyloric antrum within one inch of the pylorus, but without involving the pylorus, should be considered malignant until proved to be otherwise, and proof of the absence of malignancy in such lesions is obtained only by serial section and careful microscopic examination."

You will note that we were careful to state that the lesion must be chronic, indurated and ulcerating, as well as occurring within the pyloric antrum within one inch of the pylorus. And, we further qualify this statement by calling attention to the difficulties in determining the accurate location of these lesions due to foreshortening of the stomach shadow; and to the danger of confusing this lesion with ulcerations extending into the antrum from either the pyloric ring or the lesser curvature.

George W. Holmes, M.D.

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RACIAL TRENDS OF THE NEGRO AND WHITE IN CERTAIN SURGICAL DISEASES*

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NEW ORLEANS

IN 1790 the negroes in the United States formed almost 20 per cent of the total population. Although they have doubled in numbers in the last half century, and are continuing to increase, they now form not quite 10 per cent of the population, for the increment is at a diminishing rate and is not in proportion to the white increase. The idea is no longer tenable, therefore, that the negro may crowd the Caucasian out of the country. On the other hand, the idea, at one time rather prevalent, that the negro might be eliminated entirely in the struggle for existence, is equally untenable now, for the negro deathrate has steadily diminished in recent years.

In slavery days the negro deathrate was probably no higher than the white, and sometimes lower, for the unsanitary couditions of life affected both races equally, and the master and slave, even though susceptible to different illnesses, suffered alike from epidemic diseases. Immediately after the Civil War, when the negro was thrown upon his own resources, the racial mortality turned sharply upward, and for a time it seemed that he was a biologic product who could survive in the environment of civilization only under the protecting care of the white man. Every indication pointed to his unfitness to survive by his own efforts. With the turn of the century, however, the negro deathrate began to fall. Formerly 35 to 40 per thousand, it is now approximately 18 per thousand, and that, as Dublin points out, is as low as the rate was in many countries of Europe before the World War.

But that is not the whole picture. The negro deathrate of 18 per thousand is an enormous improvement over what it formerly was, but it is still almost twice as high as the white rate of 9.9 per thousand, and it is generally admitted that in matters of health the negro race is fully thirty years behind the white race. The negro span of life is decidedly less. At birth the negro male has a life expectancy of only 40.5 years, against a white male expectancy of 54.1 years, and the negro female has a life expectancy of only 42.3 years, against a white female expectancy of 56.4 years.

Other circumstances further complicate the picture. The negro may furnish only one-tenth of the total population of the United States, but 85 per cent of that proportion is congregated in the South. Cities such as New York, Chicago, Detroit and St. Louis, all of them outside the South, now have negro populations of more than 100,000 each, but the negroes of New York, for instance, are lost among its teeming millions, while approximately the same number in New Orleans form roughly 25 per cent of its population.

The negro in the South, furthermore, is a different person from the negro in the North. For more than two centuries he lived in slavery—it is significant that negro slaves were landed at Jamestown a year before the Pilgrim Fathers landed on Plymouth Rock—and for the three-quarters of a century that have elapsed since his

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emancipation, he has lived in virtual servitude. In the South the negro is traditionally the servant of the white man, and there are no immediate indications that the relationship will change. His life is very different from what it was in his native Africa or on the plantations of the pre-War era. He is now living in an environment for which he was not originally bred. He has lost his native strength and accentuated his native weakness through repeated intermarriage and by the intermixture of white blood introduced by direct miscegenation. He has an inherent capacity for idleness and a shiftless good nature based on the firm belief that his "white folks" will look after him.

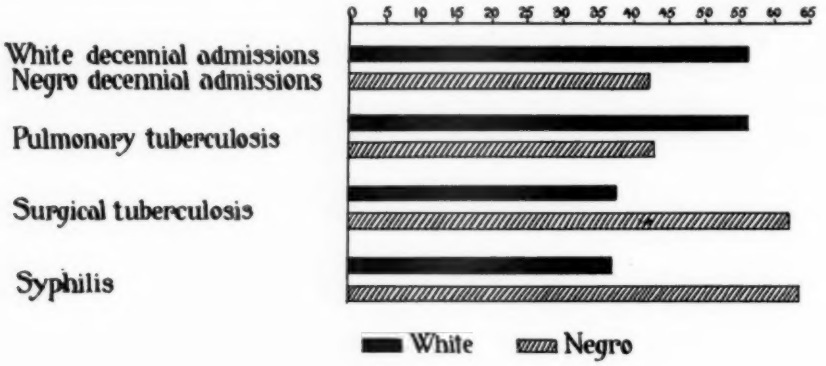

Chart 1.

And his "white folks" do look after him but not altogether from humanitarian motives, it must be admitted. As life is lived in the South, the white man could not ignore the negro even if he would. Social relations between the races do not exist, and yet, paradoxically, the relationship between them could scarcely be closer. The negro cooks for the white man, serves his meals, cleans his house, launders his clothes and often makes them, nurses his children, indeed, spends most of his waking hours in his home, and often lives in it altogether. Since, as Embree puts it, bacteria do not recognize a color line and ignore segregation edicts, it follows that the health of the negro is a matter of vital concern to the white population.

It is even more a matter of concern to the white physician, whose responsibility it largely is. The negro is a burden which he must assume, usually without thought of financial return, and as he knows from long and sad experience, one likely to cause him untold trouble and anxiety.

The negro is a real medical problem; he has an incredible disregard of the simplest law of hygiene and he does not adapt himself well to city life. In slave days the negro woman represented the very zenith of human reproductive ability, but today, perhaps fortunately, her natural fecundity is decidedly less, and the mortality of newborn infants is a national disgrace. The negro may have lost his susceptibility to his native diseases but he has developed a sensitivity to the diseases of civilization and they have proved disastrous to him. Even today, as Dublin points out, one negro in every six still dies of tuberculosis, and the same writer estimates that if this single disease could be controlled more than five years could be added to the negro's span of life. The facilities in the New Orleans Charity Hospital for the care of negroes with this disease are less than half those of the white, but the negro incidence (Chart 1) is entirely out of proportion to the facilities, and the deathrate is many times higher than in the white population, 23.7 per cent to 39.1 per cent for pulmonary tuberculosis, 5.1 per cent to 9.8 per cent for all other varieties.

The negro is also the victim of all sorts of social diseases. Syphilis (Chart 1) works havoc in both its direct and indirect mani-

festations. Its cardiovascular and cardiorenal complications appear at a relatively early age and frequently make him a very poor surgical risk. In fully 50 per cent of all cases on our negro male service operation must be deferred until syphilis and its complications have been treated, and a large part of the deathrate on that ward can be attributed to luetic disease.

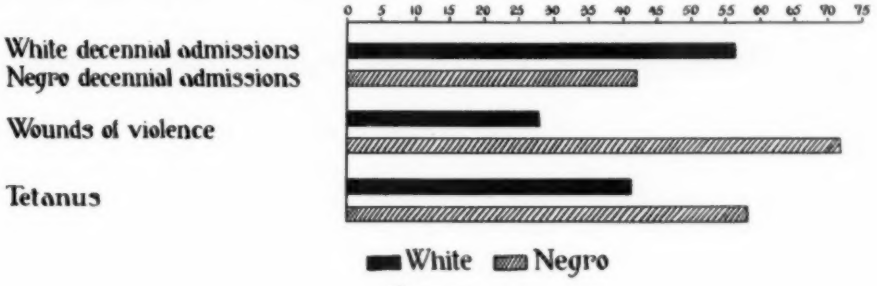
0 5 10 15 20 25 30 35 40 45 50 55 60 65 70 75
White decennial admissions
Negro decennial admissions
Wounds of violence
Tetanus
White
Negro

Chart 2.

A study made by C. Jeff Miller from Charity Hospital in 1928 corroborates the high incidence of social disease in the negro. Pelvic inflammatory disease, he states, is frequently of incredible severity and is roughly twice as frequent in the negro woman as in the white. His opinion that at least 90 per cent of it is specific in origin is really conservative, for the high incidence of gonorrhea in the negro male is notorious, as is the general racial tendency to promiscuous sexual relations.

Hookworm and pellagra, although they are now fairly well under control in the white population, still appear, often in severe forms, in the negro, as do malaria and typhoid. Deaths from violence are from five to nine times higher in the negro than in the white, the records (Chart 2) showing that more than two-thirds of the gunshot and stab wounds handled in Charity Hospital over the last ten years occurred in the negro. Boland's experience on the negro division of the Grady Hospital in Atlanta corroborates our own, and while Connors and Stenbuck's report of chest wounds from the Harlem Hospital in New York does not emphasize race, we found it quite characteristic that the single patient pictured should have been a husky negro male.

It was equally characteristic, too, that nineteen of the twenty-one human bites treated in Charity Hospital in the last ten years should have been in negroes. It is an old plantation tradition that "the bite of a blue-gummed nigger is pizen," and the surgeon who treats colored patients knows that that is true, though he attributes the poison, more scientifically, to the presence of the spirochete of Vincent.

It is frequently denied by scientists and by scientific statisticians that the mulatto differs in any way from his black or white brothers, but surgeons who operate on them know that while the pure black negro is the best surgical risk in the world, the mulatto is the worst, and that the risk increases in direct proportion to the pallor of his skin. The negro may decline surgery through fear of it, but if once he accepts it, probably because his perceptions are duller and his sensibilities less keen than those of the white man, he regards it and its possible consequences either with a complete lack of interest or with a certain pleasurable excitement, for the negro loves above all things to hold the center of the stage. These facts, combined with his inborn habit of submission to his white superiors, make him an excellent subject for any sort of anesthesia, though in occasional instances an emotional negro undergoing general anesthesia may exhibit a marked stage of excitement. Our experience corroborates that of Royster, that the negro does not pass easily into surgical shock, but that it is extremely difficult to get him out of it when once it has occurred. His postoperative reaction, case for case, is less marked than that of

the white patient, again, we would say, because of a difference in his response to physical and psychic stimuli.

The most disastrous characteristic of the negro, from the surgical point of view, is his tendency to disregard inaugural symptoms of disease. He does not seek medical aid in any illness until pain is a symptom. Since his perception of pain, as we have just indicated, is rather dull, and since pain is not an inaugural symptom of all illnesses, the surgeon frequently does not see him until the disease is advanced quite far. Miller, in discussing fibroid tumors in the negro woman, makes the same point. That type of tumor is at least nine times as frequent in the negro woman as in the white, and yet, so slow is the negro in seeking medical advice because the condition is not associated with pain until it is advanced quite far, fully 50 per cent of these tumors can be palpated abdominally when the patients are first seen, and fully 30 per cent of them are visible on casual inspection.

Tetanus is another excellent illustration of the results of this habit of delay. In the last ten years at Charity Hospital there were 343 cases of tetanus in the negro (Chart 2) well over half of the total number, while the negro deathrate was 56.4 per cent against the white rate of 54.1 per cent. During the last five years, however, the differences are even more striking, the negro mortality then being 65.1 per cent against 54.5 per cent for the whites. The discrepancy, we think, is quite easily explained. Even ignorant white patients are coming more and more to seek medical care for minor injuries, and the administration of tetanus antitoxin s saving them from worse consequences. But the negro ignores his original injury, and, since pain is not a feature, he also ignores the initial symptoms of tetanus, seeking hospitalization only when the disease is so far advanced that treatment does not always avail.

Of necessity the negro has occupied for a long time the attention of the white man. As early as 1811 a "Professional Planter" had a little manual published in London entitled "Practical rules for the management and medical treatment of negro slaves in sugar colonies." The regret voiced by the planter, that in spite of the large negro population of the country, few persons had devoted their attention to negro diseases, was echoed forty years later by a certain Dr. S. A. Cartwright who served as the chairman of a special committee appointed by the Medical Association of Louisiana to study this subject. His report in 1851 "on the diseases and physical peculiarities of the negro race," is incredibly mediaeval in its tone. The negro, he says, is the direct descendant of Ham; his nerves and blood are as black as his skin; he is born to slavery, unfitted for any other sort of life and chained to it, among other reasons, by his habit of sleeping with his face covered, which prevents the proper "atmospherization" of the blood.

The negro suffers from a peculiar form of pneumonia, Cartwright states further, which is not due to eating dirt, as was then generally believed, nor is it located in the lungs, as a modern physician might hastily surmise. It arises, instead, in the mind, and is due to mismanagement on the part of the master and superstition on the part of the slave. A disease known as drapetomania or dysaesthesia Aethiopis is discussed at length. It is defined as "hebetude of mind and obtuse sensibility of body," but overseers, in a simpler and blunter fashion, are in the habit of calling it rascality. This disease, says Cartwright, proves conclusively the wisdom of slavery and the remedy for it is hard work. The same medical treatment, continues the author, which would benefit or cure a white man might kill a negro, and it is safer for an overseer to treat negroes empirically than for the medical advisers of the Queen of England to treat them, since these physicians are ignorant of the comparative anatomy and physiology of the race.

Dr. Cartwright, naturally, did not have it all his own way. A good many physicians took exception to his report, including a certain Dr. J. T. Smith, who said flatly that if Dr. Cartwright declined to locate pneumonia in the lungs, he should devise a name for it which would signify his own ideas of its origin. Dr. Cartwright's favorite disease, drapetomania, he would have none of, hinting that the good doctor had invented it himself. It is a curious commentary on the times that all the papers in this symposium deal rather with the question of the origin of the negro race and of the advantages and disadvantages of slavery than with medical facts and theories.

From this time onward occasional discussions of the surgical diseases of the negro appear in the medical literature. Quite typical is the paper read in 1887 by Dr. L. McL. Tiffany, Professor of Surgery in the University of Maryland, before the American Surgical Association, and equally typical is the discussion which followed it. The majority of surgical conditions discussed are relatively unimportant today, and most of the major surgical states which occupy the attention of modern surgeons are scarcely mentioned, but many of the conclusions are still valid. Certain surgical affections, these surgeons decided, pursue different courses in the negro and the white man under identical hygienic surroundings. Surgical injuries and operations are better borne by negro than by white patients, because their stolid courage and moderate postoperative constitutional disturbance make them excellent subjects for surgery. They recover more rapidly than white patients because their native indifference prevents them from entering fully into the conception of surgical procedures. But these statements do not apply to the mulatto, who is a poor risk and does not bear shock well.

Not precisely germane to the subject, but interesting to surgeons of the new era, is the bold statement of Dr. E. H. Gregory of St. Louis, that he personally believed that pus always implied infection, and that as a matter of convenience, whether it be true or not, he personally accepted the idea that all suppuration of a strumous character was due to a microbe. Equally interesting to surgeons associated with the New Orleans Charity Hospital today, where much the same conditions still prevail, is the statement of Dr. T. G. Richardson of that institution, that in the out patient department "the number of patients is so great that we simply prescribe for them and let them come on a ticket."

In 1898 Rudolph Matas published a comprehensive study of the surgical statistics of Charity Hospital for the ten years ending in 1894. "The surgical peculiarities of the American negro" is still a very valuable contribution, though, like Tiffany's, it does not include the major problems of surgery today. There are only 34 cases of appendicitis, for instance, in the whole decennium, 25 in whites with 7 deaths and 9 in negroes with 2 deaths. The frequency of syphilis and the resulting frequency of its visceral and cardiovascular complications in the negro are emphasized, as is the greater frequency and higher mortality of pulmonary tuberculosis and other respiratory infections in the negro. Circulatory disease is twice as frequent and more than twice as fatal in that race. Venereal disease is more prevalent and three times more fatal. Only 43 cases of renal calculus are noted in the whole decennium, only 10 of which are in the negro, a tendency which continues today in an even more notable form. In the ten years ending in 1933, the negro proportion of all varieties of urinary calculi is only 14.3 per cent.

It might be well to pause at this point and note that the New Orleans Charity Hospital, an institution now more than one hundred years old and equipped with more than 1800 beds, furnishes opportunities for the comparative study of racial disease which probably do not exist anywhere else in the world. At the time of Matas' study something over 33 per cent

of the hospital population was negro, and the yearly admissions averaged something over 6000. In the last few years the yearly admissions have been 50,000 and more, and in the decennium which we have chosen to study, 1924–1933, inclusive, the ratio of white to negro admissions was 56.7 to 43.3, roughly 4 to 3.

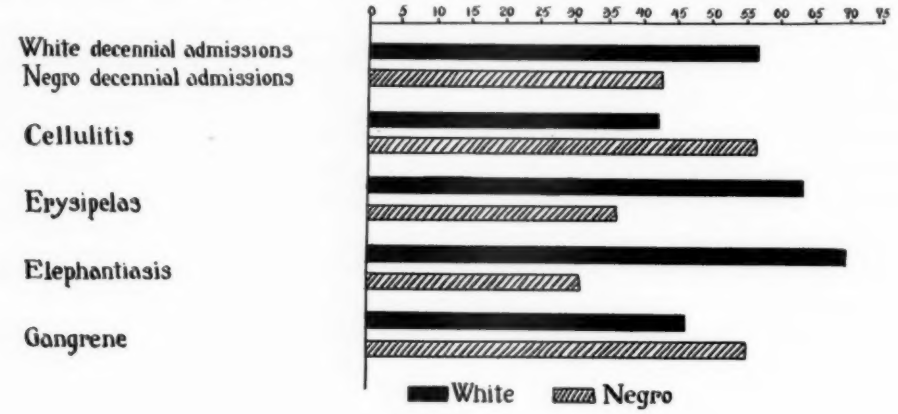

Chart 3.

It is generally admitted, as both Tiffany and Matas point out, and as our own figures prove, that certain infections are far less frequent in the negro than in the white, perhaps because his thicker, tougher, more active and more highly pigmented skin serves as both a mechanical and a chemical protection against microorganisms. Cellulitis, erysipelas and elephantiasis are all far less frequent in the negro (Chart 3) though the mortality of erysipelas is more than 6 per cent higher, probably due in part to neglect of the early lesion and to the tendency evident in the negro throughout this study for a relatively infrequent disease to show a markedly elevated mortality. Both Tiffany and Matas comment upon the apparent racial immunity of the negro to streptococcic infections, and modern statisticians support them, and note that the infrequency of scarlet fever is another example of the same tendency.

The tendency of the negro to develop vascular disease is particularly notable. In the decennium under consideration there were 741 cases of various sorts of non-traumatic gangrene, more than half of which (Chart 3) occurred in the negro. The predominance is not difficult to explain. The negro is frequently syphilitic, is an early victim of arteriosclerosis, quite as susceptible to diabetes, contrary to the general belief, as is the white man and he is far more likely to ignore its initial manifestations. Then his pedal hygiene is bad, he wears rough, ill-fitting shoes that are full of holes, and his socks, if he wears any, are in like condition. Finally, he does not wash his feet very often, and partly because of the dirt and partly because of his natural color, he is prone to overlook minor abrasions and injuries, while if he discovers them, his first instinct is to smear them with some sort of greasy salve.

The mortality of gangrene in the negro is higher than in the white, being 32.6 per cent against 28.4 per cent, and he tends to develop this complication at an earlier age than does the white man. J. Ross Veal has recently pointed out in a study of amputations for gangrene in Charity Hospital that the white arteriosclerotic develops his gangrene at 66.7 years, but the negro at 63.7 years, while the white diabetic develops his gangrene at 64.5 years but the negro at 51.9 years. The negro incidence of gangrene in diabetes, Veal states in the same study, is 13 per cent, against only 7.5 per cent in the white race.

Matas' report does not separate the various forms of abdominal hernia, but his collected statistics show that the condition

is more frequent in the negro than in the white. In our own study, in which the different varieties are considered separately, the figures are exactly reversed (Chart 4) except for umbilical hernia, which is probab.y more frequent in the negro than in the white as the result of carelessness in the management of the cord at birth. Certainly the prevalence in the white race, which, at least in this climate, does not do the hard manual labor performed by both male and female negroes, would seem a convincing reply to those who champion the traumatic origin of hernia.

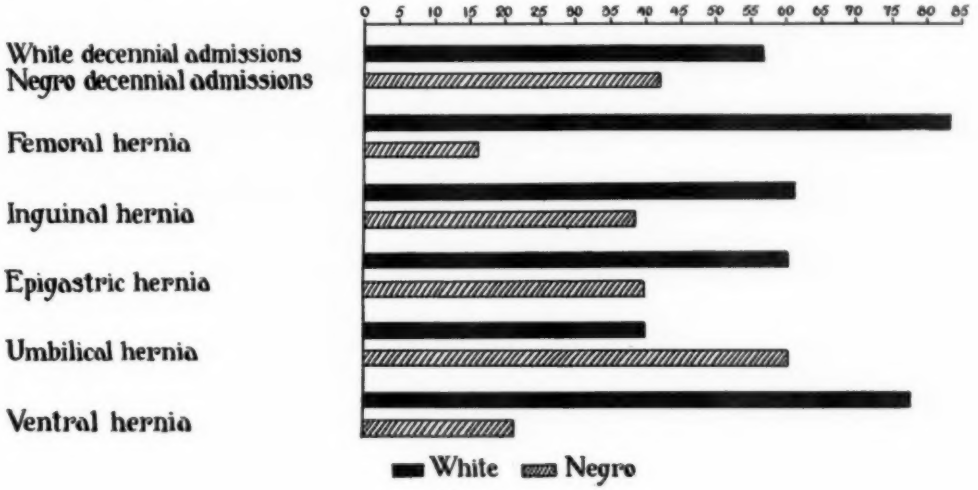

Chart 4.

The percentage of strangulated hernia in both races is almost in proportion to the hospital population (Chart 5), but in intestinal obstruction due to other causes the majority of cases are in the negro. This is in line with other reports, notably those of Boland and Finney, and Finney's explanation is logical, that the higher incidence in the negro is due to the higher incidence of pelvic disease in negro women. Indeed, it is remarkable that intestinal obstruction from postoperative adhesions is not more frequent in negro women, in view of the severity of the disease in this race and the difficulty of achieving proper peritonealization after the pelvic organs have been removed. Boland's theory is at least as good as any other, that such results as are achieved are due to a saving grace which prevents these women from going very much farther. We agree with him, too, that there is no evidence to support the idea that because of the tendency of the negro to keloid formation, peritoneal adhesions and bands are more likely to develop after trauma and inflammation.

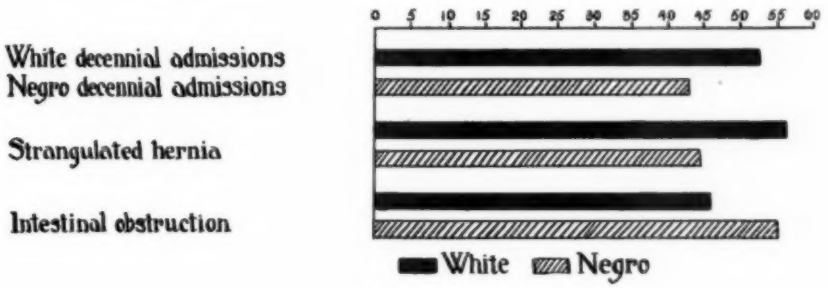

Chart 5.

Boland comments, as we must also, on the danger in intestinal obstruction in the negro of the violent peristalsis induced by the huge doses of salts and "black draught" which they are in the habit of taking. The

peristalsis augments the swelling and edema at the site of the obstruction and may cause a partial block of the bowel to become complete. Undoubtedly the thing that keeps the negro mortality in intestinal obstruction as low as it is, 50.8 per cent against 49.9 per cent in the white, is the continuous and violent character of the pain, which forces him to seek relief much sooner than he otherwise would.

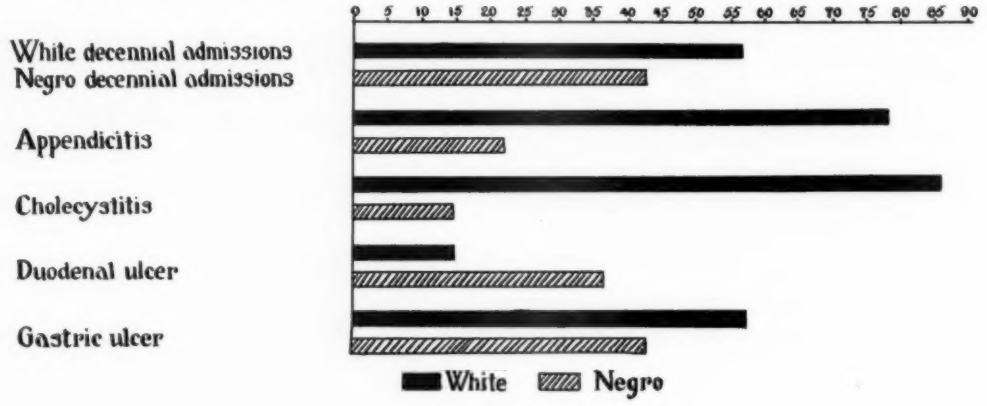

Chart 6.

The subject of purgation leads naturally to appendicitis, in which disease its damage is outstanding, and in which the relative incidence of the disease and its relative mortality in the negro and the white are particularly striking. In the ten year period under consideration the negro furnished less than one-quarter of the 4157 cases of acute appendicitis (Chart 6), though he furnished more than one-third of the deaths, and the negro deathrate was more than 7 points higher than the white, 13.4 per cent to 6.1 per cent. Part of the mortality is undoubtedly to be explained by the negro's delay in seeking medical attention and by his habit of repeated, drastic purgation, but that explanation is not enough. Taking appendicitis case for case, the negro, ill the same length of time as the white patient, tends to exhibit a severer and more advanced type of disease, and the only explanation is that the manifestations of this disease in a race which was once immune to it are exceedingly grave when the natural immunity is lost. Harbin, Miller, Boland and others have commented on this same point. The negro's loss of immunity to appendicitis may have something to do with his change in diet; support is given to that theory by the fact that when both races are given the same food in the army, the negro incidence of appendicitis is practically that of the white soldiers.

To continue with diseases of the digestive tract, the incidence of gall-bladder disease in the negro is small (Chart 6), as is the percentage of gallstones. Why cholelithiasis should be so infrequent, especially in the negro male, is not clear, though all writers on the subject, notably Jaffe, Alden, Bloch and Boland, stress the fact. Emile Bloch, who a few years ago studied all the cases from Charity Hospital, could arrive at no explanation other than a true racial immunity. Diet, in his opinion, was not a deciding factor, though Boland attributes considerable importance to it. Bloch pointed out that while the outdoor life and active physical occupations of the negro probably caused less bile stagnation than in the white patient, and while the absence of constipation in the negro may play some part in his immunity, these factors would seem to be overbalanced by the greater frequency of pregnancy in the negro woman, for pregnancy produces bile stagnation and hypercholesteremia. The infrequency of biliary stones in the negro is clearly related to the infrequency of urinary stones, upon which we have already commented.

As in appendicitis, the mortality for biliary surgery is considerably higher in the negro than in the white, 13 per cent against 10.1 per cent, and the tendency seems to be general, for Boland's recent report on gall-bladder disease in the negro gives a mortality of 14 per cent. It is interesting to note that the negro, like the white man, may develop the so-called "liver-kidney syndrome" after biliary surgery. This subject has recently been thoroughly investigated by F. F. Boyce, who reports that 3 of the 23 deaths of this sort collected from the Charity Hospital records occurred in negroes.

Equally puzzling problems are presented by the statistics for other diseases of the digestive tract. Both duodenal and gastric ulcers are less frequent in the negro than in the white, although the incidence is increasing and neither condition is any longer, as it once was, a surgical curiosity. As Lemann comments, the negro ought not to have peptic ulcer at all, if Crile's theory of the kinetic drive be correct, whereas the incidence of the disease is too high to make that theory tenable. Boland's idea is probably correct, that changing food habits are responsible for the increasing frequency of this and other digestive diseases in the negro, but that is not all of the story; it is quite possible that the strain of modern life may be beginning to affect the negro just as it has affected the white man. That there is no true racial immunity to peptic ulcer seems to be proved by such a report as that of Bergsma from Abyssinia, in which he comments on the relative frequency of the disease in these primitive negroes, and attributes it, very logically, to the pepper sauce which forms a large part of their diet from the time they are weaned.

Later studies have not supported the earlier idea that sarcoma is the most frequent manifestation of malignancy in the negro; in our own study its incidence in both races is almost in proportion to the racial distribution of the hospital population. In cancer, on the other hand, very striking differences are apparent for different locations, and it is unfortunate that the early reports of Matas and Tiffany do not permit such a comparison. In both of their reports all varieties of cancer are discussed under one heading, and Richardson's specification, in the discussion of Tiffany's paper, that he was talking of cancer "in the general acceptation of the term" makes one doubt whether the diagnosis was exact in all the cases.

Both of these early reports mention the infrequency of carcinoma of the face and oral cavity in the negro, and that tendency still prevails. In the last decennium in the Charity Hospital less than 10 per cent (Chart 7) of all the cancers of the cheek, nose, lip, mouth and tongue occurred in the negro, and no adequate explanation is available for this marked discrepancy. The pigmented, thick epidermis of the negro may explain his immunity to carcinoma of the oral cavity, which is lined with mucous membrane. As a matter of fact, if the theory of irritation holds, that type of cancer should be very frequent in the negro, for he smokes very dirty pipes and his oral hygiene is usually unspeakable.

The incidence of cancer in the negro ought, logically, to be considerably less than is its incidence in the white race, for cancer is a disease of middle and old age, and the negro, with his shorter life span, has fewer individuals in the higher age groups. That is not the case, however. Negro women in the childbearing years, Miller states, tend to develop cancer of the genital organs more frequently than white women, and the same tendency is observed in cancer of the breast, in which the negro and white incidence in relation to hospital admissions is practically reversed (Chart 7). Whether the development of malignancy is related to previous disease of the breast it is not possible to say, but it is a curious coincidence that the relative proportions of breast abscess and similar conditions in the negro and white hospital population precisely parallel the figures for malignant disease, although

benign breast tumors are much less frequent in the negro. Malfunction or nonfunction of the breast gland does not explain malignancy of the breast in the negro woman, as it is sometimes supposed to explain it in the white woman, for the negro mother who does not nurse her baby is a rarity, and she nurses him long after a white woman would have weaned him.

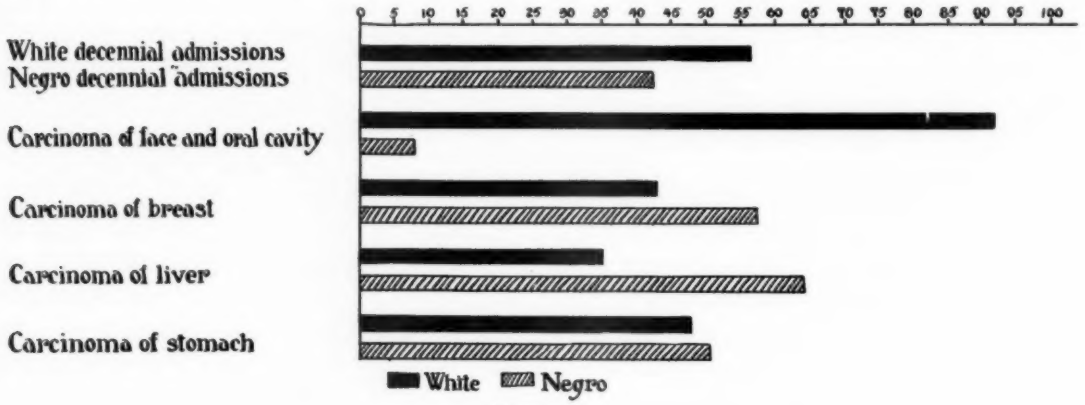
0 5 10 15 20 25 30 35 40 45 50 55 60 65 70 75 80 85 90 95 100
White decennial admissions
Negro decennial admissions
Carcinoma of face and oral cavity
Carcinoma of breast
Carcinoma of liver
Carcinoma of stomach
White
Negro

Chart 7.

Since carcinoma of the liver (Chart 7) is the most common variety of malignancy in primitive peoples, it is not surprising to find that 18 of the 28 cases recorded in the Charity Hospital records occurred in the negro, this group including the only 2 cases in the female. It is generally supposed that syphilis, malaria, typhoid, alcoholism, opium addiction, cirrhosis of the liver and similar conditions are predisposing factors, and if they are, the negro predominance is easily explained, for one or more of these factors was apparent in 14 of the 18 negro cases.

Carcinoma of the esophagus occurs about in relationship to the ratio of hospital admissions in both races, but carcinoma of the stomach is considerably more frequent in the negro (Chart 7), and at a much earlier age. In a series of 200 cases, about evenly divided between the races, which we studied in detail, we noted that almost half of the cases in the negro developed between thirty and fifty years of age, whereas only a little more than a quarter of the cases in the white developed in the same age period. The negro with carcinoma of the stomach is usually in a deplorable state when he seeks medical aid. He does not come to the hospital until definite obstruction is present or pain is a pronounced feature; the inaugural symptoms, which are more insidious in this disease than in almost any other, he ignores entirely. That fact perhaps explains why the white mortality is rather higher than the negro; bad as is the white patient's condition, it is at least better than the negro's, and more radical surgery, with a higher potential and actual mortality, is therefore attempted on him.

The incidence of carcinoma of the intestine does not differ materially in the two races, but the distribution of carcinoma of the rectum (Chart 8) is rather difficult to understand. If the theory of chronic irritation holds, it would seem, again, that the negro incidence should be higher than the white, whereas it is really considerably less. With the single exception of hemorrhoids, every benign disease of the rectum, all of them introducing factors of chronic irritation, is more frequent in the negro than in the white. The remarkably high incidence of rectal stricture in the negro is worthy of special comment, in view of the recent investigations by Lichtenstein in the Charity Hospital, which prove conclusively that the great majority of rectal strictures of inflammatory origin are due to lymphogranuloma inguinale, rather than to syphilis, as was so long supposed. As the result of his study,

Lichtenstein concluded that this condition was the most frequent manifestation of lymphogranuloma inguinale in the negro the prostate and penis, but its infrequency in the bladder is still left unexplained. The conclusion here, as elsewhere, is that

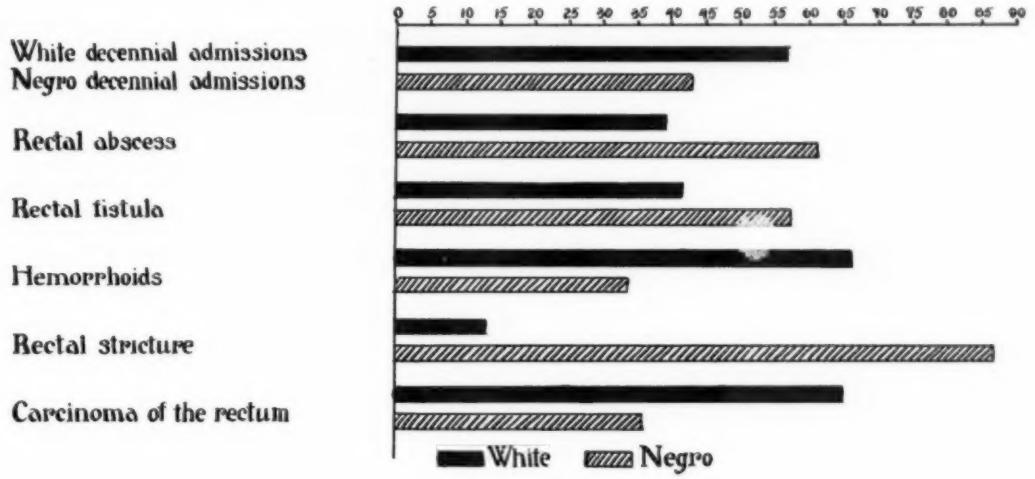

Chart 8.

female, and his necropsy observation of 6 cases with previous Frei positive reactions has enabled him to describe a clearer and more complete pathologic picture of this type of stricture than any hitherto published.

The infrequency of hemorrhoids in the negro, to which Matas originally called attention, is also worth special comment. Lichtenstein has pointed out that the rubbery anal tags which are frequent in rectal stricture due to lymphogranuloma inguinale are frequently diagnosed as hemorrhoids though they are not really varices, and it is quite possible that many of the cases diagnosed in the years before the Frei test for lymphogranuloma inguinale was introduced at the Charity Hospital, are really part of the picture of rectal stricture and are not hemorrhoids at all.

Inequalities of distribution are noted throughout the cancer statistics. Malignancy of the bladder, for instance, is disproportionately frequent in the white hospital population, cancer of the prostate and penis in the negro. The frequency of veneral disease in the negro and his abuse of the sexual act may have something to do with the incidence of malignancy in the distribution of malignant disease in the negro is a matter of racial tendencies, for which no adequate explanation has yet been adduced.

Respiratory diseases, as we have already noted, are more frequent and take a heavier toll in the negro than in the white race. The discrepancy evident in pulmonary tuberculosis is equally evident in pneumonia, influenza and empyema (Chart 9) and is particularly notable in empyema. In proportion to the hospital pneumonia admissions the negro incidence of empyema is more than three times lower than the white, though the mortality is more than two and a half times as high. Quite evidently the negro, although he seems to develop empyema less frequently than the white man, is very much less capable of withstanding its ravages. A detailed study of the records has revealed two other curious facts, that for some reason negro children are singularly resistant to empyema, and that for some reason the negro male, in proportion to the total number of cases in that race, is singularly prone to develop the disease and to develop it in a fatal form.

There is a rather general belief that thyroid disease is rare in the negro and

that the toxic variety is practically never seen. That this is untrue our figures (Chart 10) clearly prove. Goiter is not endemic in Louisiana, but the decennium figures reveal 613 cases of all varieties of thyroid disease, the negro ratio of 44.3 per cent being slightly higher than the ratio of negro hospital admissions. The toxic variety, as a detailed study of a special series shows, furnishes about half of the negro cases, the nodular variety being slightly more frequent than the diffuse variety.

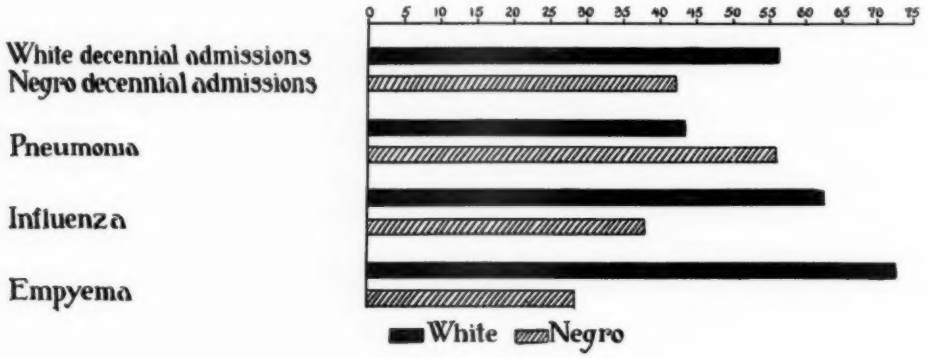

Chart 9.

stage is reached. The average negro woman does not present herself for treatment as the average white woman would, partly because her sensibilities are less keen, partly because her toxic manifestations are less marked, and chiefly because thyroid disease causes no pain. In time involutionary changes occur and the acute toxic stage passes into the nontoxic nodular stage. Whether or not toxic manifestations appear again depends upon whether or not the factors responsible for the original toxicity again become operative. Finally, in the occasional case, it is quite possible that the gland may return to its original colloid or resting state by the working of the law that all thyroid disease will eventually "burn itself out" if the patient lives long enough. A study of the individual histories makes this theory seem perfectly logical, for they prove that the negro woman carries her thyroid enlargement and endures her symptoms longer than the white woman before she seeks relief, which would explain the preponderance of nodular goiter in the

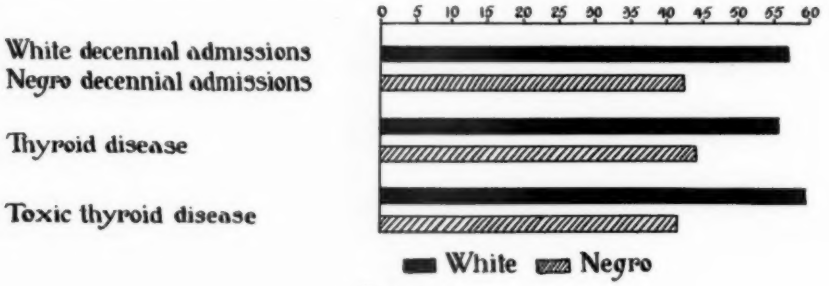

Chart 10.

It is our opinion, on the basis of our own observations, that toxic thyroid disease arises in the negro just as it frequently does in regions where goiter is endemic, on the basis of a previous nontoxic diffuse goiter, this variety being considerably more frequent in negro females than is generally realized; it is overlooked because it rarely gives rise to symptoms. In the occasional case, as the result of psychic trauma or some similar event in a peculiarly susceptible individual, who is, we believe, usually a mulatto, the inactive gland is fanned into activity and the diffuse toxic

race, as well as the rather large number of colloid goiters noted in middle-aged negro women.

The mortality of thyroid disease in a non-endemic area is always higher than in an endemic area, which explains why the general mortality for such surgery in the Charity Hospital is 6.1 per cent. That fact does not explain, however, the marked differences between the negro and white mortality for all forms of thyroid disease and especially for the toxic variety, 12.3 per cent against 9.6 per cent in the white, nor does it explain why the mortality for negro males should be 25 per cent, 15 points higher than the mortality for white males.

Part of the explanation undoubtedly is the negro's tendency to delay medical consultation in any disease in which pain does not force him into the hospital, but other factors are also operative. He may have passed safely through a series of mild crises, but they are bound to have left their mark upon him. He frequently exhibits myocardial disease and extreme degrees of hypertension, which his syphilitic tendencies have helped to produce. Furthermore, the technical difficulties of thyroidectomy in the negro are much greater than in the white, because the musculature of the negro neck, in both sexes, is extraordinarily well developed. Finally, the glands are frequently large and adherent, distorting and compressing the trachea and causing at operation respiratory difficulties of the gravest sort.

The most disturbing consideration of toxic thyroid disease in the negro is the marked increase in incidence in the last four years, the increase being more pronounced than in the white, and being quite out of proportion to the hospital admissions. The inescapable conclusion is that the negro is losing his natural placidity amid the increasing difficulties of modern life and particularly in the financial turmoil of the last few years, though as yet there is no evidence that there is associated with this change any assumption on his part of any responsibility for himself and his own welfare.

SUMMARY

From this rather superficial and casual survey of certain important surgical diseases as they are exhibited in the negro race, one or two outstanding facts emerge. The negro has an apparent racial immunity to certain infections, chiefly of the streptococcic variety, to certain forms of malignancy, chiefly of the superficial variety, and to biliary and urinary lithiasis. He is less likely to develop such diseases as appendicitis, cholecystitis and peptic ulcer, but more apt to exhibit them in a graver form than does the white man. He is peculiarly susceptible to respiratory infections, and even in such diseases as empyema, in which the incidence is low, the mortality is prone to be very high. The inescapable conclusion is that as the negro loses his immunity to the so-called acquired diseases or diseases of civilization, he tends to exhibit them in a virulent form, and that the virulence is aggravated, always, by his mode of life, his disregard of inaugural symptoms and his pernicious habit of self medication.

To the surgeon who treats negroes such a study as this is considerably more than a mere analysis of figures. Each group of statistics recalls to his mind the negroes he has treated for that disease, and so often has treated in vain. Whatever the reason, fundamental or environmental, the records of the Charity Hospital clearly prove that the negro responds to certain diseases in a different fashion from the white man and usually in a more disastrous fashion. Every analysis of statistics from this institution ought to be made on the double basis of the negro and white hospital population, for the surgeon who operates on negroes, especially for acute or grave diseases, most of the time begins his work with a very heavy handicap.

[*For References see p. 20.*]

TWO STAGE AMPUTATION FOR DIABETIC GANGRENE OF LEG

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WHEN a major amputation of the lower extremity becomes necessary for diabetic gangrene, the type of operation which will give the greatest assurance of life is the operation of choice. Occasionally this might be a formal operation with flaps and immediate closure of the tissues and then the amputation site and the treatment of the bone end can be chosen so as to be aids in locomotion with an artificial leg. However, in the greatest number of patients, concern as to the type of stump which will be most serviceable in walking should not influence the surgeon's plans, at least, such concern should not be a directing force in the primary operative procedure. Some of the patients will never need an artificial member; death will determine that problem for them. And the death rate for the treatment of this gangrene is already too high, due to the practice of formal and completed amputation in one operation.

The amputation in these patients has for its object the exclusion of infected and toxic material. This complication of diabetes usually starts with a small patch of gangrene or an infection. Later the infected area becomes gangrenous or the gangrenous area becomes infected. As absorption of necrotic products, toxins and bacteria proceed, there is an aggravation of the diabetes, and, in turn, the local condition becomes worse with the constitutional change. Consequently, it seems plain that the surgeon's task is to plan for immediate control of the local process.

When the surgeon compounds his problem, adding to his objective either the purpose of an end-bearing stump, or the plan for primary healing, he will reduce the chance of that patient's recovery, and also reduce it to a degree proportionate with the complexity of the operation. He will see, and not infrequently, the flaps he fashioned so painstakingly change color, slough and retract. The wound so clean and fresh at the time of closure will become gray and pus will ooze from its edges. Thus, the old condition for which the operation was done is reborn with all the attendant hazards. The life that was to be saved is again endangered and often lost.

The deathrate from the one-stage operation, the flap operation and immediate closure, is unnecessarily high. In a group of 56 such operations within the past ten years at the Episcopal Hospital, there were 27 deaths, nearly 50 per cent mortality. Nine of the deaths occurred within a few days of the operation, and might be accounted as the result of improper preparation. Removing these from consideration there would still be a 38 per cent mortality associated with the formal amputation. That deathrate is worse than other reported series but the best figures are far from impressive. Ralli and Standard recorded 33 per cent mortality; Bothe's figure was 22.5 per cent and for amputation of the thigh, McKittrick and Root record a 17 per cent mortality.

The deathrate in our series is traceable to the operation itself. In the 47 who survived the operation long enough for an estimation of primary healing there were 36 patients who developed gangrene or infection of the stump; in 29 there was distinct gangrene of the flaps and of these there were 17 deaths, a 59 per cent mortality. All of the 47 amputations, excepting 4, were done above the knee so that the

gangrene was not due to the selection of an amputation level which furnished the poorest blood supply. The gangrene, the infection and the 59 per cent mortality ensuing from the gangrene are an arraignment of the method of formal amputation in diabetes.

Reviewing the high mortality and the 76 per cent occurrence of wound complications, the explanation for these poor results seems obvious. Beyond doubt the irreducible minimum in deathrate will always be high because the patient is primarily a poor risk. Also, the figures will vary in communities depending upon the class of patients that is served by the hospital. But, above these factors which are fixed, more or less, the relationship of the local pathological process to the surgical procedure is a determining factor in the deaths and the sloughing wounds. The entire affected extremity is potentially infected for living bacteria are frequently found in the fascial planes and lymphatics. Furthermore, the gangrene escries the fact that the blood supply is poor, that the vascular channels are choked. Every change seen in the limb warns one that there is danger for any procedure which will open spaces or disturb circulation. It requires a group of cases, such as those I have recorded, to make one read the danger signals.

Formal amputation for diabetic gangrene is fundamentally wrong. It involves, in the making of the flaps, dissection of one layer of tissue from another at the expense of some blood supply, since vessels passing from deeper to superficial structure are divided in the maneuver. In the diabetic, all the blood supply is sorely needed and the portion destroyed may be the element which induces gangrene of flap edges. There are other factors in that complication, such as tight sutures, short flaps, etc., but the disturbance of the vascular bed is not the least.

The larger the flap the more readily will complications occur. The sloughing edges should be more common in the use of one long flap with a short one, than in equal sized flaps. In the former, there is a relatively large dissection and consequent interruption of blood vascular distribution to the skin surface. The long flap and the thick flap provide devitalized tissue for the action of bacteria. Whether the infection comes from without or from within (the contamination results from the tissues as often as from errors in technique), whichever route the tissues of the diabetic resist poorly the bacteria. When there are flaps in tiers, as in circular amputations, there are recesses for the incubation of the infection. It is not surprising that primary healing after flap amputation in the diabetic is the occasional occurrence. It is a tribute to the patient that one-half of them survived the secondary infection in the stump.

The greatest assurance for the life of the patient can be secured by the guillotine or Einschmitt operation. This does not mean the modified flap operation, leaving the stump open, for that provides only for some drainage in case of infection; it does not exclude damage to circulation nor does it give the maximum amount of drainage. All the tissues should be cut at one level and preferably it should be done in the leg about two-hands breadth below the tibial tubercle. Performed in this fashion there is little disturbance of the blood supply and small chance of gangrene, provided a tourniquet is not used. Also, the stump is wide open and without recesses, with a minimum of devitalized tissue, in fact, it has everything that discourages suppuration.

Of course, secondary operation will be necessary to give a serviceable stump. This I usually do after several weeks, or when bacteria counts of the wound show only two or three in a field. Between operations the wound is dressed at weekly intervals with cod liver oil until clean enough to start bacterial counts; then dressings are done every other day.

The secondary operation might take any form the surgeon may choose; a Carden or even a Stokes-Gritti may be done with

safety, I believe, for the reason that the removal of the foot and portions of the leg has permitted the development of a good collateral circulation. Removal of the foot and leg also provides a more rapid circuit of blood and a better nourishment of the tissues.

I have been content, at the second operation, with excising the scar and the projecting bone, cutting the fibula higher than the tibia and sawing the bone ends high enough to permit closure of the soft parts without tension. Whether this will be the final method, I am unable to forecast since pressure sores from the artificial limb may cause a change in the plans. However, unless there should be a persistence of the ulceration, I prefer a plan that will save the knee joint at the expense of an occasional ulcer of the stump.

SUMMARY

In summary, the simplest amputation that provides drainage and undisturbed vascular distribution is offered as a means of controlling the deathrate in diabetic gangrene.

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*Continued from p. 17.

IDIOPATHIC BENIGN HYPERTROPHIC PYLORITIS

(BILLROTH HYPERTROPHY)

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GASTROENTEROLOGY is indebted to Cruveilhier for a large number of original observations and descriptions of pathological changes in the region of the pylorus and duodenum. In 1835, Cruveilhier described the postmortem findings in a woman of seventy-five years, who had died from pernicious vomiting, and recorded essentially the same findings that one would describe today, in a condition which he named adult pyloric hypertrophy. This condition he designated as an inflammatory condition of a peculiar type, involving only the pylorus, with hypertrophic features superimposed in certain of the layers. His description so many years ago corresponds quite accurately with any present description of the same lesion.

Many other clinicians confirmed Cruveilhier's findings and similar cases were reported to 1890, when Billroth stressed its existence as a distinct disease entity so vehemently that many of his assistants designated it "Billroth Hypertrophy."

It is possible that in Billroth's development of the Type I and Type II gastric resections bearing his name, he encountered cases resected for a supposed carcinoma of the pylorus based on the gross findings, but since the expected malignant changes in the specimen were not present microscopically, his attention was diverted more acutely to the condition, as many other surgeons since his time.

The term generally accepted as descriptive of this entity is idiopathic pyloric stenosis, and the feature stressed by a large number is that there is a hypertrophy of the muscular layers in the region of the pylorus producing a tumor like formation with induration and obliterations of the pyloric canal. A review of the literature on the subject produces considerable confusion and perplexion, so that any attempt to coordinate existing accurate information regarding the lesion with personal clinical experience in an endeavor to throw more light upon it can be pardoned.

For a number of years many pathologists have stated that a lesion which could accurately be called a hypertrophic gastritis did not exist. In more recent years, however, various men, more particularly Konjetsny, have studied resected gastric material and postmortem specimens which have shown that this premise is not true. Clinically also, various procedures have come into use which have added additional corroborative evidence that true chronic gastritis does exist, such as, for example, the technique of Berg for roentgenologically demonstrating the relief outline of the mucus membrane of the stomach. More recently intragastric photography and the increased amount of intragastric observation made possible through the introduction of the flexible gastroscope, has served to furnish additional evidence that the thesis that a true hypertrophic gastritis did not or could not exist was untenable.

In the description of the clinical entity to be given here, namely, benign hypertrophic pyloritis, it is not intended to convey the impression that this condition is a simple acute or chronic inflammatory gastritis with the well known accompanying mucosal changes. It is desired to emphasize that the lesion is individualistic, in that the changes described are peculiar to it alone and are confined to one sharply localized area in the stomach only, namely the

pylorus. Also that the most predominant feature is hypertrophy, confined mainly to the mucosa and submucosa, with only occasionally some extension downward into the muscular layer of a fine network of new fibres and cell infiltration but is not to be confused with a hypertrophy of the muscular layer per se. The mucosal layer shows distinct enlargement and marked thickening, causing such an increase in the size of the mucosal folds that they encroach upon the lumen of the pylorus and interfere with egress from the stomach so that secondary stasis of either moderate or extreme degree may take place. Also these enlarged ruga in the pyloric region may run in all directions and cause a complete occlusion of the pyloric canal, if the degree of change is considerable.

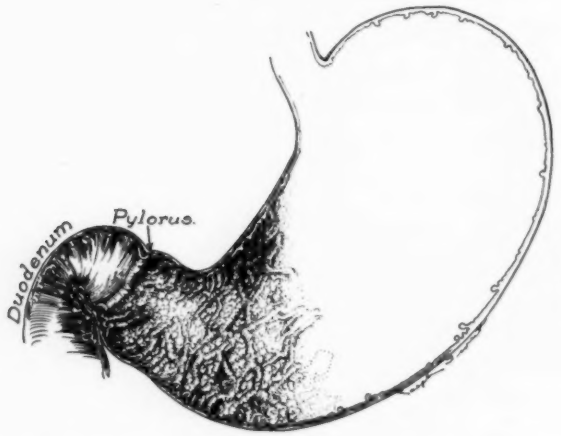

Fig. 1. Schematic drawing illustrating arrangement and size of ruga in region of pylorus of normal stomach.

One of the difficulties encountered in endeavoring to demonstrate the lesion is that it is best shown in the living subject, where the edema and congestion is readily apparent to both inspection and palpation, but this opportunity is only rarely possible. The edema and congestion rapidly diminishes or completely disappears after death so that postmortem sections are not of the greatest value for demonstration purposes.

When encountered surgically, on opening the abdomen it is found to be fairly soft and somewhat yielding, in contrast to a linitis plastica or a malignancy which is usually hard, resistant and non-yielding.

A curious point, which may be of some value in aiding to recognize the condition and first called to attention in 1933 by Cole in an article on hypertrophic gastritis, is that although it is normal for the ruga in the pyloric region of the stomach to run in all directions, that is, longitudinally, obliquely or even transversely, there is a tendency in this particular entity especially for the folds to run more obliquely and transversely to the long axis of the stomach than longitudinally. If this observation can be checked in a large series of cases, then it can be used diagnostically in questionable cases. By using the Berg technique of demonstrating in relief the gastric folds during fluoroscopy and rechecking with films, sufficient information may be obtained, invaluable in differential diagnosis. The usual normal tendency in the pylorus is predominance of longitudinal ruga.

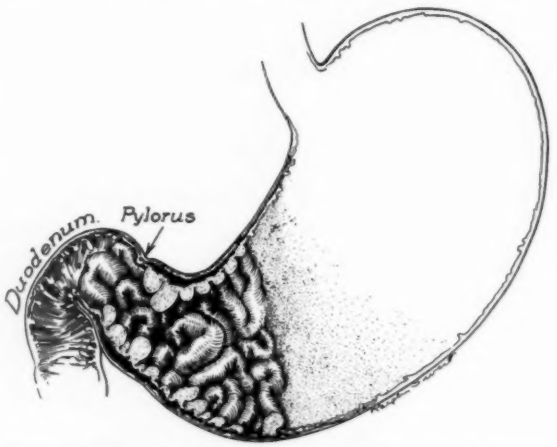

Fig. 2. Schematic drawing illustrating the increase in size, number and mass of rugal tissue in benign hypertrophic pyloritis.

After the condition has been present for a considerable length of time and a gradually increasing amount of pyloric obstruction has been produced, due to the mechanical interference with the outlet of the stomach by the hypertrophied mucosal folds, secondary hyperperistalsis may take place in an effort to overcome the obstruction, thereby possibly producing a compensatory hypertrophy of the muscular layer, a work hypertrophy so to speak.

In histological sections of this lesion allowance must be made for technical difficulties in their making, and in a way

(d) The number of tubules is increased numerically thus adding to the size and bulk of the region.

Fig. 3. Photomicrograph illustrating the microscopical changes found in this lesion as enumerated in the paragraphs describing pathological findings.

Fig. 4. Photomicrograph illustrating the microscopic appearance of the individual villi under high power, emhasizing the increased amount of cell infiltration

the sections distort somewhat the true picture of the lesion.

Dehydration and fixation of the specimen tends to reduce their size and swelling, thereby the actual thickness of layers. Blood vessels and lymph channels, which in the true picture are distended, become diminished or contracted in size. Primarily, however, the microscopic picture can be said to demonstrate the following:

(a) The mucosal layer is markedly thickened over normal and thrown into increased number of folds.

(b) In the mucosal and submucosal layers, the latter particularly, there is an increased number of blood vessels which are over distended and filled with blood corpuscles.

(c) There are increased intra- and extracellular infiltration of lymphocytes and monocytic type cells, with increased size of the individual tubules, so that the entire picture is one of over presence of cells.

(e) There is an infiltrative fibrolastic cell stroma which tends to form a basement membrane and which in some cases may dip down into the muscular layer as a loose stroma, with again the tendency to increase size and bulk.

(f) An increased amount of lymphoid cells and lymphoid tissue are found.

As stated by Klose and Bernstein the hypertrophy is due to an increase in the mass of the individual fibres in the mucosa as the other layers show no histological changes. The mucosa is folded in all directions and thus secondarily narrows the pyloric lumen, and then may invaginate to a greater or less extent into the duodenum. The picture, however, must not be confused with one of normal large sized ruga in this region, particularly along the greater curvature, or confused with that of a general hypertrophic gastritis in which the ruga are enlarged and hypertrophied in a consider-

able area of the stomach extending for a considerable distance upward toward the cardia. In the specific condition described here, there is a sharp line of demarcation and the hyperplastic picture limits itself definitely only to the pylorus. It must also not be confused with a picture of exaggerated size of normal ruga in which the cell infiltration found in the mucosa and submucosa is entirely absent.

It is rare that a true organic stenosis occurs in these cases, because even with a high degree of rugal hypertrophy, to the point of actual penetration and invagination of the hypertrophied folds through the pyloric ring into the duodenum, there is usually a rapid passage of the food column into the duodenum because of the soft compressibility of the tissue in contrast to the rigid unyielding tissue of a lesion involving deeper layers, particularly the muscularis, such as is found in a linitis plastica or a malignancy. It is because of this yielding of the tissue that Kirklin's sign, of which we shall speak later under roentgen aspects, is produced. Therefore the absence of immediate gastric retention in these cases may constitute another point of clinical value in differential diagnosis. Secondary retention produced because of the mere bulk of food added to the bulk of hypertrophied ruga could normally be expected. Also secondary spasm of the stomach in the area just proximal to the involved area might occur as the result of the stimulus to hyperperistalsis on the part of the stomach in an endeavor to rid itself of its load.

ETIOLOGY

Several theories have been advanced as to possible etiology. Among these the following have been more prominent:

(a) *Congenital.* The condition represents a continuance of a congenital pyloric stenosis into adult life. The adherents to this theory maintain that a history of gastric difficulties in infancy or in early childhood can be obtained. The evidence against this is that congenital pyloric stenosis represents an actual hypertrophy of the wall of the pylorus involving all the layers, particularly the muscularis, and the lumen of the pyloric canal is actually narrowed, at times to complete occlusion. In actual stenosis there are no changes in the superficial layers such as are described here.

The term "idiopathic pyloric stenosis" which has been suggested by some authors for this entity is a misnomer as no actual structural stenosis exists. This is a point which needs to be stressed.

(b) *Acquired.* Another theory is that the condition is secondary to a long maintained spasm of the pyloric sphincter, such as may occur as a reflex in the case of a long continued active ulcer, either presently active or long since healed.

If this were true one would expect to obtain a rather suggestive ulcer history on some of these cases, which clinically we are unable to obtain. Secondly one would expect that the hypertrophic changes would be primarily in the muscular layer, a real compensatory work hypertrophy of muscle, but this is not so, as the changes are in the superficial layers, the muscular layer showing either very little or no change whatever. In 1915, Truesdale observed that in cases of ulcer with pylorospasm, when ready egress from the stomach was established by gastroenterostomy, the musculature of the pylorus underwent atrophy. It is reasonable to presume that with an increased load and a persistent back pressure continued over a long period of time, the opposite effect or a hypertrophy, could be produced. However, as already stated hypertrophy of the muscle layer is not a feature of this condition and is only rarely present and then in moderate degree.

(c) *Reflex Spasm.* This has been advanced as another cause, specifically, persistent continued reflex spasm secondary to gall-bladder, colon or renal pathology. The same factors would hold true with regard to this theory as mentioned in the previous paragraph.

(d) *Endocrine.* It has been proposed that the condition may be the result of a

hyperadrenalism, based upon the observation that the secretory stimulation of the pancreas is inhibited by excessive adrenalin production and that lack of pancreatic secretion acts to delay the neutralization of the acid chyme from the stomach, thereby causing the pylorus to remain closed an abnormal length of time. It is claimed that in this manner gastric retention is produced followed by gastric hyperacidity with subsequent pylorospasm, then pyloric hypertrophy. This theory might apply, as mentioned previously, if the changes found were in the muscular layer.

(e) *Vegetative nervous system imbalance* results in the production of long continued pylorospasm with secondary hypertrophic changes. This might occur as part of the picture of a hyperthyroidism, but the same reasons applied against the previous theory hold here also.

(f) *Lesion of the Plexus Myogastricus.* One German observer, Holsti, claims to have found histological evidence of inflammatory changes in the cells of the plexus myogastricus situated in the deeper mucosal and muscular layers. He claims that these changes might be infectious in origin.

CLINICAL SYMPTOMATOLOGY

The clinical symptoms of idiopathic hypertrophic pyloritis may closely simulate either ulcer or carcinoma. The following symptoms have been more prominent in the cases seen personally and are listed in the order of their clinical frequency:

1. Epigastric fullness and distress and a sense of heaviness, usually occurred after ingesting a small amount of food, so that, in some cases the amount of food intake is limited.

2. Epigastric distention and bloating with considerable belching of gas.

3. Weight loss is usually attributable to the diminished caloric intake, but may become quite alarming and confuse the picture when a differential diagnosis between hypertrophic pyloritis and carcinoma is to be made.

4. Pain may be related definitely to meals, occurring an hour post prandially, but rarely of the hunger or empty feeling type and usually described as a fullness or a tugging sensation, frequently relieved by food intake. It is not a particularly prominent symptom in any of the cases seen personally.

5. Vomiting usually without nausea is not generally present at the onset of symptoms, is often self induced for the relief of symptoms.

6. Constipation possibly is due to the change in the dietary intake.

7. Anemia, secondary in type, not severe and not usually due to any blood loss although it is possible for some superficial ulceration and bleeding to take place, occurs more as a part of the picture of decreasing weight and asthenia. Superficial ulcerations can be induced by mechanical maceration of the crowded folds in the pyloric ring, particularly if foreign matter, as coarse fibres, etc., form a part of the bulk mass and are squeezed and macerated in the pylorus, with resultant bleeding from traumatic erosion.

8. Hemorrhage can be caused by the same mechanical factors mentioned and so account for the presence of blood in the stools and gastric contents.

DIFFERENTIAL DIAGNOSIS

It would be generally conceded that the differential diagnosis between benign and malignant lesions of the pylorus presents difficulties. The main issue which has to be decided in these cases is whether one is dealing with a pyloric malignancy or a benign hypertrophy as the two closely simulate each other, particularly in the clinical findings. The finding of occult or gross blood in the gastric contents cannot be relied upon to any great extent as a differential diagnostic point because of the possibility of trauma being the causative factor in a group of instances, as mentioned previously. However, where there occurs a strong, repeated reaction in several examinations it might be more significant of a

malignancy and a constant series of negative findings would be considered more in favor of a benign lesion. At least the repeated finding of occult blood in either gastric content or stool would serve to create such an amount of doubt that more intensive scrutiny would be undertaken with a leaning toward a diagnosis of a malignant lesion, all other findings being equal.

By this is meant that if a questionable pyloric area is demonstrated on fluoroscopy and *x*-ray, with all other factors being equal, as presence or absence of hydrochloric acid in the gastric contents, etc., and if repeated examinations of the gastric contents for blood were negative, one would hesitate; whereas, if the repeated examinations were positive one would unhesitatingly suspect malignancy rather than benign hypertrophy.

The presence of free hydrochloric acid in the gastric contents would, of course, be the only finding which could be significant but not conclusive, because we have all seen cases of carcinoma of the stomach in which free hydrochloric acid was present. The presence, however, of a marked increase in hydrochloric acid would be significant and of aid because it is a frequent finding in these cases of hypertrophic pyloritis in contradistinction to the diminished acid values or achylia found in malignancy.

Additional factors of diagnostic help necessary to make the clinical picture more complete are the *x*-ray findings. Kirklin has listed a group of pyloric conditions which roentgenologically closely simulate one another and which must be considered in differential diagnoses of pyloric lesions. A few more have been added to his list, mentioned here, without attempting to list relative frequency of occurrence, etc.

(a) Simple benign pyloric hypertrophy,
(b) Secondary hypertrophic pyloritis associated with pyloric stenosis,
(c) Simple antral spasm,
(d) Gastric syphilis,
(e) Carcinoma of the pylorus,
(f) Multiple polypi at the pylorus,
(g) Pyloric or prepyloric ulcer with marked peri-ulcerous infiltration,
(h) Circular myoma.

The possible relationship of antral spasm to the lesion is one productive of speculation. Whether long continued spasm could produce sufficient superficial irritation and inflammation in the superficial areas to be the precursor of this entity is within the realm of possibility. Specifically this would include reflex spasm secondary to ulcer, an inflamed gall bladder or the presence of gallstones, an inflamed appendix, or in association with a spastic colitis. If it were possible to prove that one or the other of these disease factors were present first and then secondary pylorospasm occurred and remained present over a long continued time, then this hypertrophic phase took place, such an observation would be exceedingly valuable but it has not been possible to do this to date. Another stumbling block is that the condition is generally found to exist quite alone without coexistent pathology elsewhere in the vicinity.

Kirklin in his discussion of the roentgen aspects of hypertrophic pyloritis cites several poignant truths which are to the point. He states, "it might be anticipated that the pyloric channel would be constantly and extremely narrowed but this is not the rule. Its diameter varies with the degree of physiologic contraction at the moment of view but in the state of relaxation the narrowing is seldom extreme, even when the hypertrophy is excessive." Cole called attention to the same phenomenon. Kirklin states further,

> . . . but contrary to reasonable assumption, obstruction sufficient to produce retention of the barium meal after six hours is found in relatively few cases, not coincidentally associated with gastric or duodenal ulcer. The reason for this is that there is no structural change, no increased rigidity, no actual narrowing or actual diminution in size of the lumen to mechanically interfere. Another feature is that secondary dilatation of the stomach is seldom encountered, as a part of or sequel of this benign hypertrophic pyloritis.

There is, however, a distinct tendency toward invagination of the mucosal folds into the duodenum. Whether this invagination occurs only during the first phase of the condition, when all change is confined to the mucosal layer alone and the deeper layer changes later, is not known. But if so, why are the symptoms present for such long periods of time and the deeper layer changes not found when these cases are inadvertently operated?

Fig. 5. Illustrating the crescentic indentation of the duodenal bulb produced by the invagination of hypertrophied folds through the pyloric ring, as described by Kirklin.

Fig. 6. Illustrating the production of Kirklin's sign.

PHYSICAL FINDINGS

Physical Examination. The objective physical signs are few. There may be some localized tenderness and spasm in the mid-epigastric region. Usually it is not possible to demonstrate the existence of any tumor mass and many authors have called attention to this point. In the cases cited no palpable mass was demonstrable.

Laboratory. Gastric analysis is important and can in many cases be of distinct help, particularly if repeated frequently and the findings are constant. Hyperacidity is the usual finding, possibly secondary to the retention in the stomach of the food particles which stimulate the production of gastric secretion. A constant, continued finding of hyperacidity is, of course, valuable in the exclusion of carcinoma as a diagnosis. Continued and repeated absence of either occult or gross blood in the gastric content is also valuable.

A lowered hemoglobin and low red blood count are usually associated with malignant lesions. Although this may occur in hypertrophic pyloritis, it is unusual and is never to the point of a frank anemia. In the cases seen there has been only a moderate lowering of the red blood count and the hemoglobin.

To reiterate, the absence of occult blood in the stool particularly in repeated examinations is of aid in diagnosis, as ordinarily we would expect continued positive blood findings in the stool in cases of malignancy. So its finding in repeated examinations is a valuable point in favor of malignancy when a differential diagnosis is being made.

ROENTGENOLOGICAL ASPECTS

Kirklin has best described the roentgenological aspects of the lesion and also the roentgenological features valuable in

the differential diagnosis. He emphasized particularly the following:

(a) A crescentic indentation making the base of the duodenal bulb concave is produced by invagination of the hypertrophied muscle. This is well demonstrated in the accompanying illustrations.

He calls attention to another important point, namely, that if the thickening were in the deeper layers producing a stiffness or rigidity of the stomach structure at this point, it would not be possible to produce this picture because the rigid structure could not invaginate. Also that during fluoroscopic examination and manipulation, by pressing the pyloric end of the indentation toward the duodenum the crescentic feature can be better demonstrated or even exaggerated. This is a most important point.

(b) Elongation of the pyloric canal, is contrary to the usual finding in either pyloric spasm, ulcer or malignancy where shortening of the pyloric canal is the rule. A second important point.

(c) The filling defect is invariably smooth and does not have the usual irregularity associated with malignancy. This, however, cannot be relied upon to any great extent as all pyloric lesions, malignant or benign can produce perplexingly similar roentgenograms.

(d) Change in the axis of the pyloric canal so that it is in an eccentric position, occurs because of the direction taken by the multitudinous folds accommodating themselves to the available space.

(e) In differentiation from ulcer, Kirklin cautions that occasionally there is a narrow crevice or rounded depression at about the midpoint of the lower border of the canal which resembles an ulcer niche, but which will not hold barium. This is because it represents only a sulcus in the folds of mucus membrane, or rather between the folds.

ROENTGENOLOGICAL FINDINGS

In our own experience it has been found that manipulation of the pyloric end of the stomach with only a small amount of barium in the stomach has been of help in determining the outline direction and physical aspects of the mucus membrane folds, whether they are soft yielding, running in one direction only or are rigid, unyielding, in stellate formation and running in abnormal directions.

Evaluating the amount of stasis present as compared to the amount of apparently hypertrophied structure is important. Only moderate stasis is present usually because of the soft yielding structure in contrast with the expected amount of stasis present in a rigid, non-yielding lesion with true structural occlusion.

The soft, yielding compressibility of the membrane folds enabling the barium to pass quite readily into the duodenum is quite an important observation.

DISCUSSION

The number of reported cases of benign hypertrophic pyloritis represents perhaps only a very small percentage of the actual number of cases. In addition there is confusion regarding this particular entity, some authors describe a "pyloritis" or "hypertrophic pyloritis" and then describe a rigid muscular induration or a tumor mass, yet in most of these cases when the microscopical picture is presented attention is called to the marked mucosal and submucosal changes, with absence of muscle layer change.

Some of the foreign surgical clinics have reported 2 to 10 cases each, as examples, Bernstein, 8 cases; Haudek, 8 cases; Wanke, 2 cases, and many others. Payr during 1924–25 resected 5 cases in which a preoperative diagnosis of pyloric carcinoma had been made and in which the resected specimen showed no evidence of malignancy but did show those gross and microscopic changes described herein as benign hypertrophic pyloritis.

In the surgical clinic at Oslo, Hanssen describes 5 cases between 1925–1931, in which a diagnosis of carcinoma was made and resection done. The resected specimen

showed grossly no malignancy; the microscopic examination revealed the described mucosal and submucosal changes and without the muscle layer changes.

Denis in a review of 20 gastrectomies which he did for clinically diagnosed carcinoma of the stomach, found 4 cases in which there was no microscopic evidence of malignancy in the resected specimen. In these cases the clinical signs were those of pyloric stenosis due to tumor, but the microscopical picture was again that of mucosal and submucosal change such as found in hypertrophic pyloritis.

Microscopically the predominant features were a proliferating element of the plasmocytic type. The mucosa was intact or covered with small papillomatous appearing proliferations. The mucosa and submucosa showed an infiltration of lymphocytic cells together with a large cell, fibroblastic in type. There was also an accompanying atrophy of the secretory glands but no increase in the amount of muscle fibre.

One could question this description of the findings and speculate as to whether or not what are described as papillomatous proliferations are not simply the increased number and thickening of the folds throwing them into such anatomical formation that they might give a superficial impression of papillomata, particularly as the microscopic picture is so consistent with that of hypertrophic pyloritis.

Two English writers describe in the same manner a case in which there was a marked digestive disturbance accompanied by a considerable loss in weight in which a clinical and roentgenological diagnosis of malignancy was made, and in which at operation the stomach was found to be enlarged and the wall thickened, with a firm tumor the size of a golf ball at the pylorus. Their description of the gross specimen was

(a) general muscular hypertrophy as manifested by an increase in the number of the fibres and in the size of the individual fibres; (b) edema of the submucosa and mucosa; (c) vascular engorgement, dilated capillaries some of which were ruptured; (d) capillary hemorrhages.

Study of a longitudinal section showed great hypertrophy of the circular muscle which forms the tumor and that this condition exists the whole length of the pyloric canal and is confined to it.

In the microscopic description, however, the following descriptive findings are noted:

"The longitudinal muscle is very much thinner over the tumor area, that is, it is not hypertrophied and it is less than normal in width and amount." The thickness of the circular layer of muscle is given as 3 mm. and of the longitudinal layer as 1.25 mm. and it is concluded that there is muscular hypertrophy. One could hardly call this muscular hypertrophy inasmuch as the normal thickness of the circular layer of the stomach in an adult is 3 mm. and the longitudinal is 1.25 mm., so there has been actually no increase in width in this particular case.

The remainder of the description gives the clue to the actual condition present, which is as follows: "the mucus membrane is folded upon itself many times and the submucous layer is increased in amount, is loose and edematous with an increased amount of fibrous tissue present." Again the picture is presented of hypertrophic pyloritis involving the superficial layers and not of hypertrophied muscle structure.

I have cited this report in detail mainly to illustrate that the number of cases reported in the literature could be greater, except that they may have been erroneously diagnosed and perhaps, more frequently, undiagnosed.

This particular case grossly gave the impression of a muscle hypertrophy and was so described but the microscopic evidence controverted this as no muscle hypertrophy was present.

THERAPEUTIC PROCEDURES

Treatment in those cases in which the diagnosis has been made and borne out by a period of observation has been mainly

symptomatic. We have paid particular attention, however, to eradicating focal infections particularly those involving the pathology been found, reiterating the statement that coexistent pathology is rarely found.

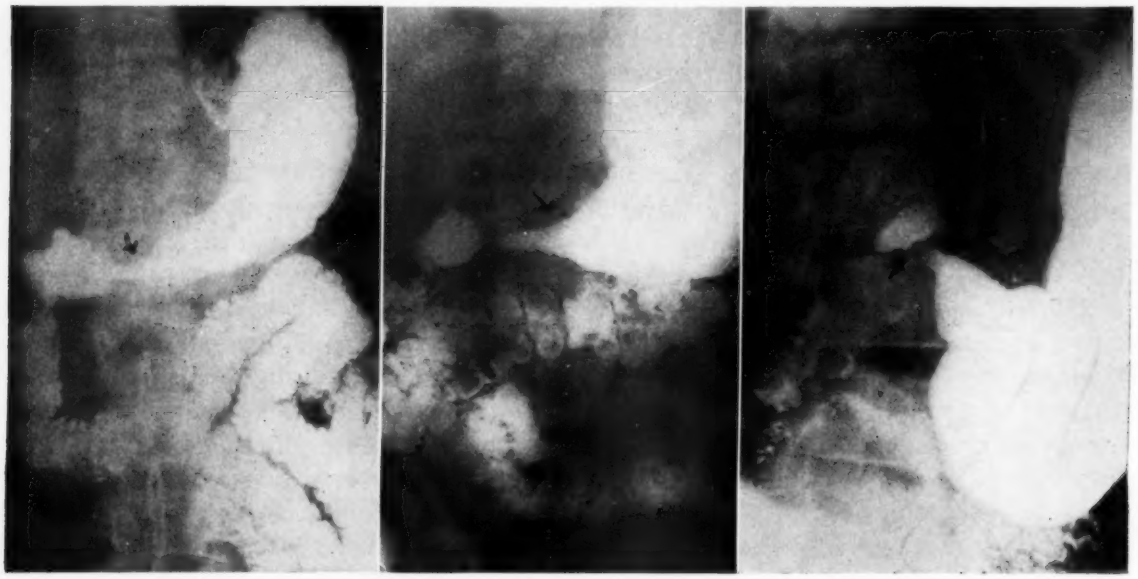

Fig. 7. Fig. 8. Fig. 9.

Figs. 7, 8 and 9. Illustrating a funnel shaped pylorus which we have found in 6 of our cases, the appearance being almost identical in each case.

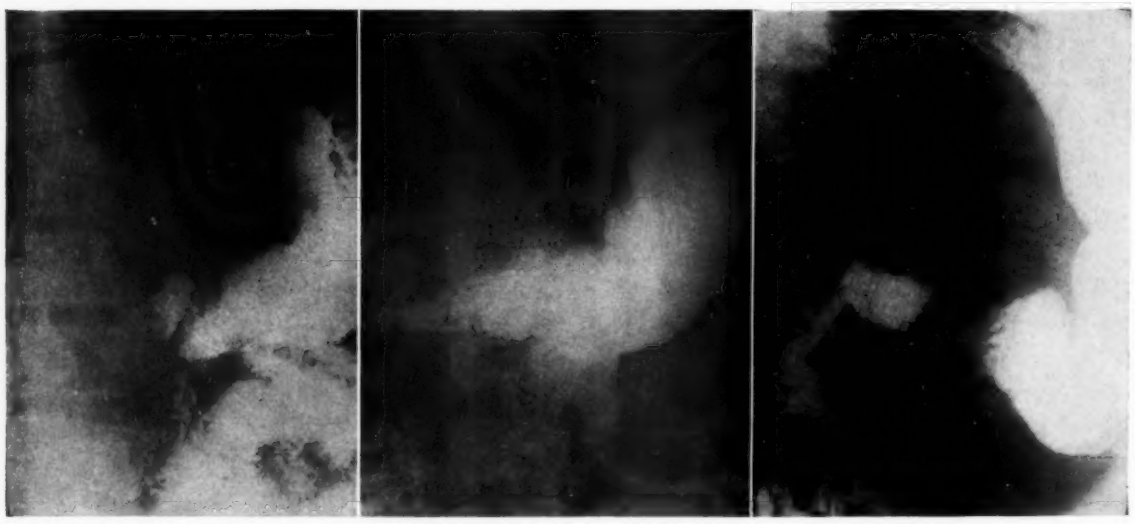

Fig. 10. Fig. 11. Fig. 12.

Figs. 10, 11 and 12. Additional pictures showing funnel shaped appearance of the pylorus of Figures 7, 8 and 9.

teeth, tonsils and sinuses. Careful investigation of these areas in all of the cases has been made and appropriate treatment instituted wherever indicated.

Gall-bladder investigations have been made in all of the cases in order to rule out the possibility of an infected gall bladder or one with stones acting as a reflex incitant to pyloric change. I might say that in none of our cases has associated gall-bladder

The dietetic procedure has been to put all of these patients upon a smooth diet, free from all roughage similar to the Alvarez smooth diet. Alcohol and tobacco have been eliminated from the patients' regime on the basis that they have irritant tendencies.

Some of the cases have been put on a modified alkalinization schedule with alkaline powders four to six times daily, using

variously the Sippy Formula in some cases and calcium carbonate or colloidal kaolin in others.

In one case the stomach was washed several times with a 1:8000 solution of silver nitrate with a rather remarkable cessation of all symptoms within a short period and no return of the subjective symptoms for a period of six months and is still symptom free. However, the *x*-ray findings six months later were unchanged from the previous ones.

In one case in which the scope of the lesion was quite large, that is the number and size of the folds was marked, a series of *x*-ray treatments had no effect upon either subjective symptoms or objective findings.

Illustrations of the radiological films in 6 of our cases are shown, with the objective of demonstrating the varying pyloric changes met thus far. Three additional pictures of the cases demonstrate the funnel shaped appearance of the pylorus as in Figures 7, 8 and 9.

CONCLUSION

1. This hypertrophic type of lesion in the pyloric region is a definite clinical entity with characteristic features entirely its own which can help to differentiate it from other pyloric lesions.

2. Due to lack of familiarity with the lesion it is frequently undiagnosed or more often diagnosed erroneously, also erroneously described in the literature.

3. Errors in diagnosis have been responsible for a fairly large number of operative procedures and particularly of gastric resections on the basis that it is a possible malignant lesion. It is believed that by taking into consideration all of the clinical findings coupled with a careful fluoroscopic and roentgenologic examination and properly evaluating these findings a differential diagnosis between malignancy and the lesion might be made and a number of cases spared the risk of an unnecessary operation.

4. It is a lesion primarily of the mucosal and submucosal layers extending the complete length of the pyloric canal but strictly confined to the pyloric region in contrast to other lesions which extend usually beyond these limits.

5. It is not the persistence of a pyloric stenosis from infancy.

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TRAUMATIC SUBDURAL HEMATOMA

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SUBDURAL hematoma is not an infrequent complication of cranial trauma but it is only in recent years that attention has been directed toward this condition. While there are more instances of head injury each year and the type of trauma does not differ to any extent, the discovery of subdural hematoma is becoming more common, probably due to earlier recognition by the physician and direction of the patient to a neurosurgical clinic.

In 1914, Trotter[14] reported 5 cases of subdural hematoma and was of the opinion that the hemorrhage resulted from a rupture of the short veins running from the cortex to the longitudinal sinus. In Grant's[7] series of 16 cases, in 10 the head was struck in either the frontal or occipital region. Thus the veins can easily be torn by a displacement of the cerebral mass subsequent to a direct blow, as emphasized by Brodie.[2] In my series of 16 cases, the blow was received in the frontal region in 7, the occipital in 6 and the parietal region in 3 instances. Rand[13] reports 3 of 7 cases in which he found a vein thrombosed within the clot and, hence, a possible source of hemorrhage. In one of Grant's cases, a vein low in the temporoparietal region, leading from the lateral sinus to the cortex, was found torn loose and embedded in the clot. Munro[11] recorded a similar case and in 4 of the author's patients a thrombosed vein was discovered in communication with the clot. The consensus of opinion is that subdural hematoma occurs chiefly over the cerebral cortex, yet in 4 cases of this series a clot has been found on the floor of the skull. However, in either location, the hemorrhage arises from the rupture of small veins in the subdural space. Gardner[6] has explained the increase in the size of the clot on an osmotic basis and as the clot is poorly vascularized, it breaks down and liquefies into fluid rich in proteins. The thin mesothelial layer of the clot, as demonstrated by Putman and Cushing,[12] acts as an osmotic membrane sucking cerebrospinal fluid into the sac. Munro confirms this view by reporting figures on the protein content of the fluid obtained from these hematomas. He feels that the gradual diminution in the protein content indicates breakdown and liquefaction with dilution of the contents of the sac by osmosis of the cerebrospinal fluid. This is not always the case because occasionally the clot remains hard and firm, making removal a tedious procedure. In all 16 of Grant's cases and in 29 of the 40 cases recorded by Jelsma,[8] the contents of the hematoma had become liquefied. This was true of 10 of my cases, however, the duration of the clot may account for a variance in this phenomenon. Because of the variability of liquefaction of the clot, with subsequent enlargement, there remains an interval between the initial trauma and recognition of this complication. Alcoholism has been mentioned as a possible cause and yet in only 3 instances in this series did alcohol play a part, one of dulled sensibility which probably predisposed to the accident. Whether alcohol exerts an irritating influence on the subdural space has not been determined definitely. In approximately 40 per cent of the cases recorded in the literature, the clot was bilateral. In this report of 16 cases, based upon operation or autopsy, the majority were liquefied clots and in 5 instances the clot was bilateral.

The symptoms will vary greatly and a definite history of injury is of extreme value. The one point that is outstanding is the bizarre clinical picture, resembling only

a disturbed cerebral function in many instances. The injury may have been trivial or of marked severity and in only 6 cases have I been able to follow each patient personally from the time of the accident to the discovery of the hematoma. Fortunately, however, accurate histories and summaries of findings have been obtained in the majority of the other cases. Contrary to many reports, 13 of these patients suffered from severe trauma and only 3 sustained a relatively mild injury. In 9 cases there was blood in the cerebrospinal fluid and fracture of the skull was demonstrated in 4 patients.

Bowen[1] mentioned a "lucid interval" existing between the injury and the development of coma, and a "latent interval" between the initial trauma and appearance of symptoms of disturbed cerebral function. In Grant's experience the lucid interval was longer than the latent interval. He also stresses the point that the advent of symptoms of hematoma is no different from any other mass in the cranium, such as tumor or abscess. In my series the shortest period was twelve days and the longest eleven months with an average interval of five and one-half months. The age incidence varied from ten months to sixty-seven years. Ten males and six females comprise this series.

Trotter spoke of insidious mental or personality changes which varied from dullness to misbehavior and finally stupor or coma. Nine of my patients demonstrated a very marked change in personality, varying from irritability to violence. One of the most constant symptoms was headache as seen in 13 patients. Convulsions were quite common as 8 patients suffered from one or more seizures. Vomiting, usually of the projectile type, was noted in 11 cases.

The objective findings were a veritable hodge-podge and in 6 cases encephalography was necessary for localization and in 2 cases ventriculography was performed. Kaplan[9] has stressed the importance of a unilateral dilatation of the pupil in determining the side of the hemorrhage. This was true in 5 cases but in 3 of these patients there was also a motor involvement of the opposite extremities. The presence of choked discs was noted in only 4 of the patients in this group.

The treatment for subdural hematoma is removal of the clot. In the past, an osteoplastic flap was turned over the suspected area and the clot was evacuated. Some authors have utilized subtemporal decompression for the purpose of approach. In 1932, Fleming and Jones[4] and McKenzie[10] suggested simple trephines placed anteriorly and posteriorly over the vault with drainage of the fluid in the cyst. Frazier[5] has used this method and Coleman[3] advocates a single trephine in the superior temporal region on each side of the skull. This method is satisfactory in some instances, but when the clot is hard and firm, a subtemporal decompression or an osteoplastic flap is necessary for complete removal.

In 8 cases an osteoplastic flap was turned, in 3 instances a subtemporal decompression was utilized, bilateral trephines were accomplished in one case and a combined trephine and subtemporal decompression was necessary in 3 cases. Bilateral trephines carry very little, if any, risk and it is my impression that they should be used in all cases to avoid overlooking a clot which may be giving no symptoms. Grant advocates a small osteoplastic flap and also removal of the dura and resuturing after turning it over to prevent subsequent oozing. In my experience, the flap has given the most satisfactory results. It has been my practice to insert a small rubber drain for twenty-four hours to facilitate further drainage. Coleman has stressed the failure of the brain to expand after being compressed by a large clot over a long period of time. This seems to be an adequate explanation for the unsatisfactory results in some instances. In addition to failure of adjustment of the brain and its circulation, I feel that softening and degeneration takes place in the

TABLE I

SUMMARY OF SIXTEEN CASES OF TRAUMATIC SUBDURAL HEMATOMA

Age	Sex	Injury	Symptoms and Findings	Operation	Results
1. J. B., 7 years	F	Blow over occiput with scalp laceration; no loss of consciousness. November, 1928.	Headache, failing vision, drowsiness and stupor. Bilateral choked discs.	Encephalography. Oct. 27, 1930. Ventriculography. Oct. 28, 1930. Removal of cerebellar clot Oct. 30, 1930.	Complete recovery.
2. E. H., 54 years	F	Fracture left temporal region. Bloody cerebrospinal fluid. Feb. 20, 1931.	Headache, vomiting, irritability; some memory loss.	Encephalography. May 28, 1931. Clot removed by left subtemporal decompression. June 1, 1931.	Complete recovery.
3. R. L., 10 months	M	Apparently a birth injury.	Convulsions, some pallor of optic discs. Slight spasticity of extremities.	Encephalography. Oct. 21, 1932. Osteoplastic flaps. Right—Oct. 24, 1932. Left—Nov. 7, 1932. Clots removed.	Fair; residual spasticity. Convulsions not as frequent but still present. Walking well.
4. J. G., 54 years	M	Blow on left frontal area. August, 1932.	Headache, vomiting, personality change, convulsions. Right hemiparsis. Aphasia.	Osteoplastic flap left frontal, July 11, 1933. Clot removed.	Fair; relieved headaches and convulsions; personality change persisted. Died, September, 1934.
5. F. B., 16 years	F	Blow on left frontal area; also laceration right arm with subsequent gas gangrene. Sept. 5, 1933.	Coma. Right hemiplegia; bilateral choked discs.	Osteoplastic flap left frontoparietal Oct. 6, 1933. Clot removed.	Complete recovery.
6. G. C. 26 years	M	Blow on right occiput Bloody cerebrospinal fluid. February, 1933.	Headache, vertigo, personality change.	Encephalography. Nov. 29, 1933. Osteoplastic flap right parietal. Dec. 5, 1933. Clot removed.	Complete recovery.
7. E. E., 24 years	F	Blow on right frontal. Bloody cerebrospinal fluid. November, 1933.	Headache, stupor. Left hemiparesis.	Osteoplastic flap right frontal Jan. 2, 1934. Clot removed.	Complete recovery.
8. H. B., 67 years	M	Blow occiput. Bloody cerebrospinal fluid. February, 1933.	Bilateral spasticity, coma, choked discs; right hemiplegia.	Osteoplastic flap left temporal Jan. 27, 1934. Clot removed. Trephine right temporal March 2, 1934. No clot on right.	Died four weeks after second operation. No autopsy.
9. F. S., 38 years	M	Blow on right temporal; fracture right temporal. Bloody cerebrospinal fluid. Jan. 19, 1934.	Convulsions. Choked discs; stupor. Left hemiplegia.	Osteoplastic flap right temporal Feb. 1, 1934. Clot removed.	Complete recovery.
10. M. V., 50 years	F	Blow on occiput. Bloody cerebrospinal fluid. May, 28, 1934.	Convulsions, aphasia. Right hemiplegia.	Osteoplastic flap left frontotemporal June 28, 1934. Clot removed.	Complete recovery.
11. C. S., 43 years	M	Slight blow over frontal region. July, 1934.	Headaches, irritable, vomiting, drowsiness.	Encephalography. Oct. 12, 1934.	Died twelve hours after encephalography. Autopsy—bilateral subdural hematoma.
12. J. B., 57 years	M	Left basal fracture. Bloody cerebrospinal fluid. May 24, 1935.	Headache, aphasia. Right hemiplegia.	Trephine, clot left temporal. July 29, 1935.	Complete recovery.
13. N. S., 50 years	M	Slight blow left frontal. June 1, 1935.	Headache, memory loss, convulsions. Choked discs.	Ventriculography. July 8, 1935. Osteoplastic flap left frontotemporal July 11, 1935. Clot removed.	Died, pneumonia third postoperative day. No autopsy.
14. J. G., 14 months	M	Blow on left frontal. Aug. 31, 1935.	Coma, convulsions. Left hemiplegia.	Right subtemporal decompression. Sept. 14, 1935. Clot removed.	Died three weeks postoperative. Autopsy—basilar meningitis.
15. B. R., 3 years	F	Fracture left frontal. Bloody cerebrospinal fluid. June 22, 1935.	Headache, vomiting personality change.	Encephalography. Nov. 13, 1935. Trephine right temporal and left subtemporal decompression. Nov. 16, 1935. Clots removed.	Complete recovery.
16. D. W., 8 years	M	Laceration left frontoparietal. Bloody cerebrospinal fluid. Oct. 29, 1935.	Coma. Right hemiplegia.	Bilateral trephine. Nov. 18, 1935. Clot removed on right; not found on left.	Died. Autopsy—basilar meningitis and clot on floor of left middle fossa.

more chronic cases. Putnam and Cushing and Munro have reported cases of edema of the brain following evacuation of the clot, but in most instances, as Coleman states, it is a failure of expansion. Usually this complication can be avoided by a decompression and the use of the rubber drain with judicious administration of fluids intravenously.

SUMMARY

Sixteen cases of traumatic subdural hematoma are presented. In 15 cases, operation was performed with 4 deaths after operation. One patient died fourteen months after removal of the clot and another died following encephalography. In 10 instances there was definite relief and 9 patients recovered completely.

A very slight injury may cause a large subdural hematoma but the majority of these patients had received a severe injury.

The history of injury accompanied by signs of disturbed cerebral function should lead to a suspicion of subdural hematoma.

Bilateral trephines should be made over the superior temporal region, followed by an osteoplastic flap and drainage after evacuation of the clot.

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THYMIC DEATH

AND METEROLOGICAL ENVIRONMENT*

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FOR the past six years it has been my privilege to investigate the causes of sudden or unexpected death occurring in adults as well as in infants and children referred to the office of the Cook County Coroner for examination. In most instances autopsies were performed on bodies of such individuals to determine the pathological anatomic causes of death. Because investigation of such cases of "sudden death" does not often come into routine hospital autopsies, the diagnosis "status thymicolymphaticus" as a possible cause of sudden death, frequently arouses differences of opinion between the hospital pathologist and the coroner's physician.

Felix Plater[1] in 1614 first described death due to suffocation by an enlarged thymus and Morgagni and Bichat associated laryngeal and acute respiratory obstruction with an enlarged thymus. Kopp in 1830 suggested the term "thymic asthma" to describe a condition with an enlarged thymus pressing on the trachea, the nerve trunk and the larger blood vessels in the neck. But Friedleben[2] in 1858 denied the association. It remained for Paltauf[3] in 1889 to suggest the term "status lymphaticus" or "status thymicolymphaticus" to describe a condition which apparently depends upon a specific constitutional anomaly of the lymphaticochlorotic type in whom resistance is markedly diminished by slight injuries, and shock and death may follow environmental alterations which would not seriously affect the so-called normal or healthy person. It was Paltauf's belief that the anatomic changes observed were only to be regarded as gross manifestations of the constitutional defect.*

As Graeme Mitchell and Brown[4] clearly demonstrate, opinions concerning the clinical disturbances attributable to the thymus have swung from one end of the pendulum to the other; the organ has been incriminated as the cause of many symptoms, but on the other hand, has been considered relatively innocent and unimportant. The truth of the matter probably lies somewhere between the two extremes, although it is not easy to know just where the pendulum should rest. Confusion is increased by the fact that certain physicians, whose convictions must be received with respect, have expressed extreme views, sometimes doubting that the thymus ever causes manifestations that will be clinically evident.

SIZE OF THYMUS GLAND

Because of the older view that the cause of death might be a mechanical one, much emphasis has been laid on the size of the thymus. Boyd[5] has investigated the weight of the thymus gland in health and in disease and her observations indicate that the thymus gland in healthy children weighs more than the thymus gland in children who have been ill. Her observations furthermore show that when illness lasted more than twenty-four hours, the weight of the thymus was reduced regardless of the cause of death, except, of course, in tumors of the thymus, leukemia, and

* Incidentally, this observation of Paltauf was made while he was medical examiner for the State (Austria).

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exophthalmic goiter. This involution is possibly part of a general involution of most organs found in inanition, but apparently the thymus, like subcutaneous fat and lymphoid tissue, reacts rapidly so that all three are significantly decreased in all diseases lasting more than twenty-four hours.

I have had occasion to corroborate Boyd's observations by removing the thymus glands of all persons who died as a result of injury. In bodies of such individuals brought to the morgue the thymus gland was removed, its size and weight recorded, and it was found that when death was due to an accidental cause, or resulted soon after injury, the thymus gland was larger in size and in weight than the glands removed from individuals previously ill for twenty-four hours or more. The size and the weight of the gland was, of course, compared to the height and weight of the body. It is interesting that Simon[6] in 1845 already referred to the thymus gland as a "barometer of nutrition, and a very delicate one" since the thymus seemed to respond more rapidly than other organs to the disturbances of nutrition.

The recent report of the British Commission who investigated the subject of "status thymicolymphaticus" has been of considerable importance in influencing the pathologists to the view that the thymus is relatively negligible. Young and Turnbull,[7] who represented the British Commission, state in their report that there is no evidence, in the relatively few data available, that acute diseases of short duration, that is, under three days, reduce the average weight of the thymus to an appreciable degree. This, of course, is not in keeping with the observation of Boyd and those that we have made in the examination of our cases of accidental death. The British investigators received their material from various parts of the country and from those individuals who were sick for a certain period of time, and not as we have indicated from our investigations, that the thymus gland was removed from individuals who died from accidental means and autopsy performed soon after death.

The British investigators further show that the normally large thymus in itself cannot be considered indicative of a status thymicolymphaticus. In most observations the cause of death can be found postmortem. To this fact there have been many subscribers, but as indicated, one does not attempt to make the thymus gland itself the only cause of death. These investigators further state that it is impossible to judge the adequacy of anesthetics or shock as causes of death because their effect cannot be measured in the dead body. It is quite true that the effects of shock cannot be measured in the dead body, but as we will show later, the individuals of the constitutional type of "status thymicolymphaticus" is very labile and responds most readily to the slightest external factors which may be responsible for releasing the various functional changes responsible for the cause of death. We cannot help but emphasize at this point that the British Commission has put too much stress on organic changes and not enough on functional and pathological physiological alterations which occur in the body. We do not possess methods for demonstrating functional clinical alterations on the autopsy table.

It is obvious that too much emphasis must not be placed on the size and weight of the thymus gland as a responsible factor in the immediate cause of death. At best the thymus probably merely mirrors the constitutional status, as Paltauf suggested, when at autopsy he observed the prominence of the lymphoid tissue and thymus in children and young adults dying suddenly with no other demonstrable cause of death. That the weight itself is of no great significance becomes apparent from the observations of Hammer[8] who found that the thymus of healthy persons dying from accidental cause within twenty-four hours of the injury, weighed as much as those of persons dying from "status thymicolymphaticus."

Clinicians, observing that children and infants who had died suddenly, frequently revealed enlarged thymus glands at autopsy, began to apply roentgen rays over the area of the thymus gland in the hope that the gland would become reduced in size and thus eliminate the life hazard. But roentgenologists have demonstrated that the roentgen findings agree with the anatomical findings in that the largest thymuses occur in healthy children killed suddenly by accident, and that the thymus weight ratio decreases with age.

In view of the fact that the mechanical explanation has been unsatisfactory, investigators have sought other factors. A disturbed endocrine dysfunction has been suggested, but the experimental evidence so far adduced has not been convincing. Of course, the complex interrelationship between the thymus gland and other glands of internal secretion has been intensively studied. Marine,[9] as well as others, has produced "thymico-hyperplasia" by the removal of the suprarenal cortex.

Non-mechanical asphyxia has been advanced as the cause of sudden death and various explanations have been given, such as factors that may suddenly decrease the oxygen carrying capacity, sudden change in the metabolism by emotion and physical exertion. It has been suggested that toxic stimulation of the heat center might account for death and that we might be dealing with a suprarenal cortical insufficiency.[10]

Recently the allergists have attempted to correlate the thymus with death and Woldbott[11] has mentioned the possibility that the thymus gland may liberate a substance, possibly a protein, which sensitizing the tissues such as the lung, may result in sudden death if at some subsequent time a shock dose of protein is liberated. This investigator suggests that this hypothesis is supported by the fact that the alveoli are dilated and that the lumen contains edematous fluid, and there may even be a rupture of the alveolar septa with thymic death. To this view I cannot subscribe, because it is the experience of most pathologists who have made histological examinations of lung tissue that this pulmonary edema is present in practically all types of deaths and may be due to a great variety of conditions. The mere presence of the pulmonary edema certainly does not imply that it is the result of an allergic condition and due to a protein or protein-like substance derived from the thymus gland.

STATUS THYMICOLYMPHATICUS: A CONSTITUTIONAL TYPE

As a result of the uncertainty concerning the mode of death the syndrome of "status lymphaticus" has resulted in the divided opinion as to the actual existence of the condition, and pathologists as well as clinicians are certainly not in agreement. In the average general hospital, or even the children's hospitals, the sudden or unexpected death in apparently healthy children is not often seen. Therefore, it is obvious that those engaged in medicolegal work will have an opportunity of observing these cases more readily than others, and we are indebted to Symmers,[12] who in a recent discussion of the subject presented the facts from this standpoint. He defines the condition as a combination of hereditary, constitutional anomalies, entering into which are certain peculiarities of configuration, with preservation or even hyperplasia of the thymus at an age when involution is to be expected; hyperplasia of the lymphoid cells of the spleen, intestine and elsewhere; changes in the distribution of hair, hypoplasia of the vascular system, developmental deficiencies in the genitalia and incidentally visceral defects of uncertain occurrence and irregular distribution. It may be of further interest to note that status lymphaticus is compatible with life, and that persons may go on to maturity with it but survive involutional changes in the lymphoid tissue. At the same time the persistence of the hyperplastic lymphoid tissue throughout the body carries with it the potential danger that the human organism may be accompanied by an

instability of the lymphoid tissue in such a fashion that it may produce various changes in the body such as has been described by investigators as anaphylactic phenomena, asthmatic attacks, convulsive seizures, or sudden death. These individuals who have the Paltauf syndrome may carry with it the added danger of the lowering of the threshold for infection, a defective development of the musculature coat of the arteries, which, in turn, may be so disturbed that the vascular system may not respond as it should under normal conditions to withstand changes in blood pressure.

Clinicians have attempted to classify status lymphaticus according to types. Some have associated the tall slender individual who is supposed to have an unusual susceptibility to tuberculosis, particularly of the lymphoid tissue, as a type that would fit into the category of status lymphaticus. After puberty and in the early adolescent period there is a type which may be associated with a delicate texture of the skin which may have a velvety touch or faintly creamy hue. The osseous system may present a slender waist and the musculature may be evenly proportioned, but with arching thighs and gracefully outlined arms. It is found that in the male the pubic hairs assume the distribution of the female and the vesical organs may be somewhat smaller than normal. This may be accompanied by a scant distribution of the axillary and thoracic hairs.

PATHOLOGIC ANATOMY

The pathologic anatomy of status thymicolymphaticus has included the lymphoid tissues, vascular apparatus, genitalia, and incidental anomalies of development of various organs. Some observers have classified the status thymicolymphaticus into two types, (1) the status lymphaticus type, in which there are changes in the new tissues at an age when these naturally flourish; and (2) the reserve status lymphaticus type in which there are atrophic changes in the lymphoid structures which may vary in extent to the time of involution and configuration which depends upon the amount of waste of the disease, and of age. It is further demonstrated that the faucial, laryngeal and pharyngeal tonsils are hyperplastic in about 50 per cent of the cases, and that the follicles in the intestinal tract are prominent in about 80 per cent of the cases. The spleen may or may not be enlarged and hyperplastic lymph follicles may be seen.

Histologically, there is an hyperplasia of the cortical lymph follicles and the blood sinuses are filled with lymphocytes. Some investigators have associated the hypoplasia of the aorta with the changes just described. Mitchell and Brown state that histologic structure of the thymus gland is difficult to correlate with function.

Some clinicians have demonstrated that patients showing a lymphocystosis of 40 per cent or over, present a dangerous operative risk and are liable to sudden death. A similar condition has been observed in Graves disease and in people with persistent and hyperplastic thymus glands. In reserve types, and those past the age of puberty, the germinal follicles often show almost complete replacements by whorl like collection of spindle cells which probably represent connective tissue elements, also large polyhedral forms resembling degenerated cells of the type of large lymphocytes.

Symmers states that the cytoplasm of both suspended and polyhedral cells stains pink with eosin, and that on this account the follicles stand out in contrast to the usual deep blue of the lymphocytes, a condition seldom seen other than in thymic deaths. Symmers suggest that the germinal follicles may release a nucleoproteid which is not strictly foreign to the body but nevertheless pathological and may be comparable in a toxicological sense to foreign products. After a certain period of incubation the lymph follicles, which are subject to further destructive changes and further disintegration of the germinal nuclei, provide the requisite quantity of

specific protein to complete this cycle. He believes that at a certain moment the tissue may become sufficiently tuned and await only the receipt of a sufficient quantity of a specific protein to react violently, even to the extent of sudden death. At other moments they might not be tuned so properly so that the quantity of specific protein which is released exerts no such effect. This explanation may or may not sound logical but Symmers believes that it may be a factor which explains why certain subjects of status lymphaticus type survive surgical procedures while others do not. The enlarged thymus and hyperplastic lymph follicles, a condition, referred to as status thymicolymphaticus, has been seen in association with cerebral hemorrhage in young infants, and those who are of an emotional instability. In fact, it is suggested that those who commit suicide, and degenerates of various types, gunmen, and other criminals who have met death by violence, show findings of a status thymicolymphaticus.

Bartel[13] and Miloslavich[14] in 232 cases of suicide report 80.5 per cent showed signs of status lymphaticus at autopsy. These changes have also been seen among epileptics and those confined in insane institutions. In persons who are emotionally instable, such as exophthalmic goiter patients, hyperplasia of the lymphoid tissue is seen. Persons who have enlarged thymus and hyperplastic lymph glands are more prone to acute infections than those who are relatively free of this syndrome.

Aldrich,[15] who has studied the symptoms of vagotonia and thymic hypertrophy, has observed that in roentgen ray treatment of the thymus gland there were many instances which suggested themselves of pylorospasm and bronchial spasm. Also that vagotonic symptoms could be produced either by stimulation of the vagus or the parasympathetics, or by reduction in the activity of the sympathetic nerve fibers. Hyperfunction of the suprarenal glands has a marked effect on the autonomic system, and that vagotonic symptoms may follow a hypofunction of the suprarenal glands.

Tracy[16] is of the opinion that death in status lymphaticus is due to lack of epinephrin hormone in the blood which makes adequate systole of the heart and blood vessels impossible. He states that in death caused by status lymphaticus the heart is always in diastole.

Aschoff[17] has proposed the view that the mechanism of the cause of death in status thymicolymphaticus is a ventricular fibrillation which leaves no pathological evidence on the autopsy table.

Marine and his coworkers have further shown that suprarenal insufficiency may be the stimulating or probably primary cause of thymic and lymphatic hypertrophy and it therefore makes possible the relationship between thymic hypertrophy and vagotonia. Mitchell and Brown further state that many of the explanations given for sudden death in status thymicolymphaticus, as the effect of an unknown toxin, "hypothymization," "lymphotoxemia," vagotonia, deficiency of the chromaffin system, and allergic reaction, are rounding in terminology but have little or nothing to uphold them. The theory of adrenal deficiency, however, has much in its favor and is supported by certain physiologic and pharmacologic facts, a discussion of which lies outside the province of this presentation. Adrenal insufficiency seems to be a possible explanation of certain cases of sudden death which can be accounted in no other way. It is interesting that in adrenalectomized animals there follows hypertrophy of the thymus. That cerebral hemorrhage is sometimes a cause of sudden death in children with enlarged thymus glands and lymphoid hypertrophy, is undoubtedly true, but cerebral hemorrhage, however, could account for only a relatively few instances of sudden death.[18]

ANALYSIS OF AUTOPSY MATERIAL

The matter of the incidence of status lymphaticus is important because one

might well question the number of death certificates bearing this diagnosis that are issued for infants and children on whom an autopsy has not been performed. In some instances, private doctors issue death certificates without performing an autopsy and it is obvious that such diagnoses as congenital heart, pneumonia, enteritis, putrid bronchitis, etc., might be questioned. In a personal communication with the pathologists at the Cook County and Michael Reese Hospitals, Dr. R. H. Jaffé and Dr. O. Saphir, both institutions have large children's hospitals, as well as Dr. Roos of the Children's Memorial Hospital; we are informed that they have no occasion to make examinations in so-called sudden death in children who have died as the result of accidental means. The incidence of status thymicolymphaticus in their records is practically nil.

During the course of the past six years I have had the opportunity of investigating a considerable number of deaths in which I made the diagnosis of "status thymicolymphaticus." The cases were all in infants and children who died suddenly without previous illness. Because of the lack of all other organic changes, the fact that the only pathological findings were hyperplasia of the lymph glands, enlarged thymus, spleen, with prominent Malpighian corpuscles, with hemorrhage either in the thymus, or in the Peyer's patches in the intestinal tract, and in the absence of significant microscopic changes or chemical evidences of poisoning, the diagnosis of "status thymicolymphaticus" was made.

In the year 1930 the series consisted of 3 females and 16 males, varying in age from one month to eight years. Eighteen of the individuals were white, one was colored.

During the year 1931 I performed autopsies on 2 white boys, three and twelve years of age, weighing 45 and 100 pounds, and being 3 feet 6 inches and 4 feet 8 inches in length, respectively.

During 1932 I had no opportunity of performing autopsies on such cases, but I have investigated the deaths so designated in the mortality records of the city of Chicago and these will be discussed later.

In 1933 an autopsy was done on a white girl six and one-half months of age, 20 pounds in weight and 25 inches in length.

In 1934 the examinations included 7 children varying in ages from two months to six and one-half years, of whom 4 were males and 3 females. In all of these groups the thymus weighed between 30 and 55 grams.

I need not point out that autopsies were made on many infants and children not included in this group, despite the fact that the general constitutional evidences of "status lymphaticus" were present in some measure, but in this group we found other changes, such as cerebral hemorrhage in the newborn, putrid bronchiolitis, and early bronchopneumonia. To avoid criticism when these two findings were associated in the same body, I prefer to make use of the latter anatomic diagnoses rather than the designation of death due to "status thymicolymphaticus."

Let us assume that an infant or child dies without any previous illness; let us further assume that the necropsy examination does not reveal any evidence of disease changes grossly or microscopically other than a somewhat enlarged thymus gland; perhaps with hemorrhages; that there is a generalized hyperplasia of the mesenteric and other lymph glands with a diffuse hyperplasia of the lymphoid follicles in the intestinal tract, which may be associated with petechial hemorrhage; that there is associated subpleural and subepicardial hemorrhage; and that there is an accompanying narrowing of the aorta. These are the only findings. Is one justified, in the absence of every other type of lesion that may be responsible for the cause of death, in assuming that what we have just described as "status thymicolymphaticus" is not the associated cause for this sudden demise of the infant?

It is further obvious that not in all deaths is one able to have anatomic proof. Are we justified therefore in assuming that

because the evidence is lacking from the pathological standpoint to account for the cause of death that the absolute proof of death is lacking? The changes which are not manifested by pathological anatomic means may be associated with altered biochemical changes, probably disturbed pathological physiological alteration; other factors such as constitutional variability which may be markedly disturbed by extrinsic agents such as physical and meteorological climaxes may hasten the onset of sudden death. In view of the fact that there is so little to account for the death as far as anatomical alterations are concerned, we must consider the possibility that we are dealing with some biochemical alteration of profound significance having its origin possibly in some unusual environmental situation.*

In work which has been done during the past decade, Petersen and his associates[19] have demonstrated the chemical and endocrine changes that are constantly occurring as the normal person reacts to the environment particularly in making the necessary adjustments to the meteorological environment. The possibility suggested itself that sudden death in the constitutional type that we have been discussing, might occur when an unusual meteorological situation might require prompt physiological adjustment.

While I shall not enter into a review of the relationships of death to the meteorological environment, I can summarize Petersen's[20] findings to the effect that with the infall of polar air, or the polar or cold front of the meteorologists, the normal person responds with a sympathicotonia. The blood pressure increases as the peripheral vascular bed contracts, the blood becomes more alkaline, the liver releases sugar, reduction is at first enhanced in the tissues, adrenal, and probably pituitary, hormones are released. Petersen has termed this the ARS* phase of the metabolic cycle. It is followed by a reversal of trends toward greater oxidation, lessening of blood pressure, lowering of the *p*H of the blood, increase in metabolism, the so-called COD phase.

With the ARS phase, or the pressor episode, Petersen has demonstrated a great variety of pathogenic expressions, the COD phase is not of pathogenic significance. If the individual with "thymicolymphatic" constitution is constitutionally deficient in the chromaffin apparatus and particularly in adrenal activity, we might anticipate that this deficiency will become acutely evident when an environmental demand is thrown upon the organism by the sudden meteorological change of the type suggested, and this might account for the acuity of the episode. In the presence

* Kolisko, (In Dittrichs Handbuch der Ärtzlichen Sachverständigentätigkeit. 2, 1913), in his classic description of sudden death due to natural causes, emphasises the importance of the lymphatic constitution, thymic death, as a constitutional anomaly responsible for the sudden death. This he observed in a large number of cases, and further calls attention to the decreased vitality persons of a lymphatic constitution have, when exposed to disease.

During the recent World War (1914–1918) Aschoff (L., Die militärärztliche Sachverständigentätigkeit. Part II, Jean 1917), performed autopsies on 250 soldiers who were in the reserve corps of the German army and who died suddenly. Of this number there were 28 cases which showed a definite lymphatic constitution.

Jaffé and Sternberg, (Jaffé, R. H., and Sternberg, H., Kriegspathologische Erfahrungen, Virch. Arch. f. Path. u. Path. Anat., 231, 416, 1921), examined 200 soldiers who died suddenly of natural causes during the World War (1914–1918). Six cases were diagnosed status thymicolymphaticus. These men varied in age from nineteen to thirty years, and the thymus weighed between 15 to 45 grams. There was an hyperplasia of the lymph glands and a narrowing of the aorta, in many specimens examined.

* The abbreviation ARS is used to designate the metabolic phase when *a*nabolism preponderates; when the blood shows a relative *a*lkalosis; when we presumably deal with an *a*drenalemia; when *r*eduction is enhanced; when *s*pasm of the smooth musculature, arterial, sphincters, etc., with general increase in the smooth muscle tonus, is apt to occur; with *s*ugar increase in the blood, etc.

The abbreviation COD is used to designate the opposite pole of the metabolic swing; is used as an abbreviation for the increase in *c*atabolism with increase in *o*xidation; with *d*ilatation of arterioles and capillaries. During this time there is a relative acid preponderance of the blood, the *p*H diminishes, the basal metabolic rate increases, the blood pressure falls, and the smooth muscle tone is diminished, it is a so-called vagus preponderance rather than a sympathicotonia.

of a definite sympathetic defect, the "vagus" factor might so far overbalance, especially in the cardiac mechanism, that

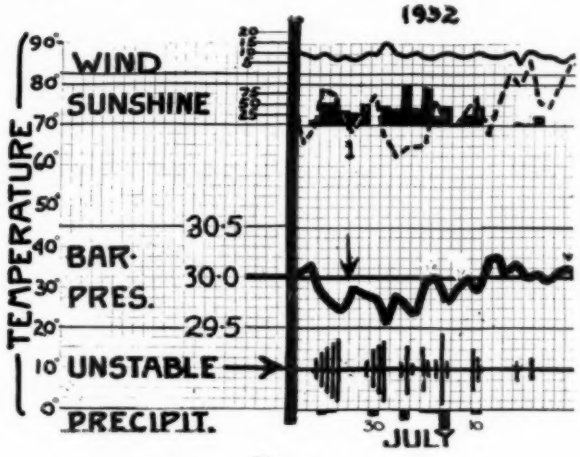

Fig 1.

cardiac inhibition might supervene, with prompt death.

It is this problem that I have sought to investigate by the metero-analysis of the periods when cases of death from "status lymphaticus" have been definitely determined.

I have prepared a series of meteorographs of which the following is an example and on these I have indicated the dates when the deaths occurred.

All of the meteorographs are from the same locality as that from which the patients are drawn. The top curve is that of the average wind velocity in miles per hour; black columns indicate the percentage of cloudiness, possible sunshine percentage being the difference. The mean daily temperature is indicated by the dashed curve, with scale at the left; the barographic curve is indicated by the heavy black line, again with scale at the left; the vertical columns below this are indicators of relative meteorological instability, not official, and the small black columns below the date line show the amount of precipitation, or rainfall.

The shifting cyclonic fronts are of course indicated both by the change in pressure and in temperature. A polar "infall" or polar "front" would be in evidence on June 27 on this small graph, indicated by the arrow 1 (part of the month of June precedes July), when cold air, denser and drier, suddenly passes over the country. With this the ARS phase, with increase in blood pressure, predominates. The cyclonic episodes that influence the human organism are the interfaces of the gaseous masses, i.e. "polar" or "tropical" fronts. With high temperature and high humidity the elimination of too much carbon dioxide, because of the increased respiratory rate, may also induce a blood pressure episode.

1930

Nineteen cases were examined by me during the year 1930, the majority occurring in the first part of the year and in definite groups, *a*, *b* and *c*.

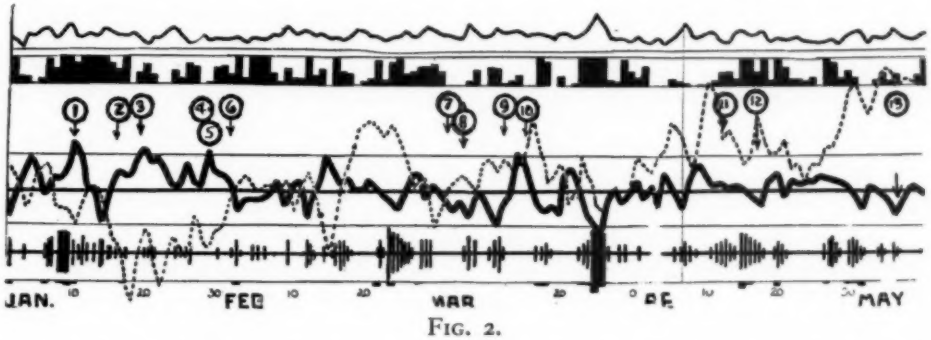

Fig. 2.

Group a. ① on January 10. This occurs at the time of barometric crest, i.e. at the maximal effect of polar air. The reader will note the characteristic combination of lowered temperature and high barometric pressure.

② and ③ occur on January 16 and 19, both with major barometric episodes in association with the coldest time of the year.

④ and ⑤ occur on January 28 and 29 on a barometric crest.

⑥ occurs on the succeeding polar infall,

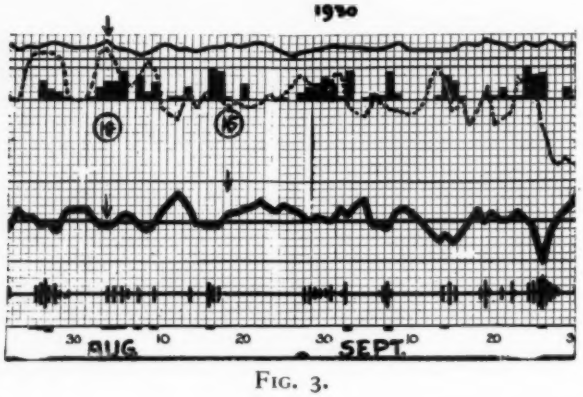

Fig. 3.

on February 1.

Group b. The next group of cases occurs with the succeeding cold wave.

⑦ on March 4.

⑧ on March 6.

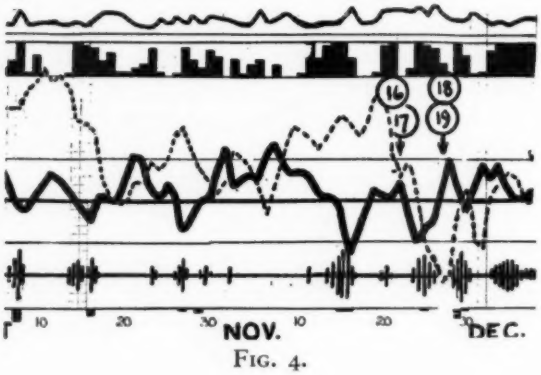

Fig. 4.

⑦ occurs in the wake of a severe polar episode, but with declining barometric pressure, but with case ⑧ a transient barometric crest is in evidence, although the weather is warm.

⑨ and ⑩ occur on March 12 and 15. These apparently occur in association with the major barometric episode of the time, ⑨ on the polar front, ⑩ immediately in the wake of the barometric crest.

Group c. ⑪ occurs on April 12 with a sudden polar infall.

⑫ on April 17 is to be related to the crest of the tropical episode. Unfortunately it is impossible from the records to determine the exact hour of death and therefore whether we were dealing with the crest of the tropical wave or whether the effective factor here was a sudden polar infall.

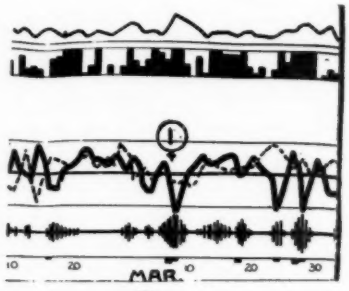

Fig. 5.

⑬ on May 7 occurs clearly on a polar front.

⑭ and ⑮ on August 3 and 18. Here again it is impossible to determine from the dating of the death in ⑭ whether or not we were dealing with the effectiveness of the unusually hot humid weather, or whether

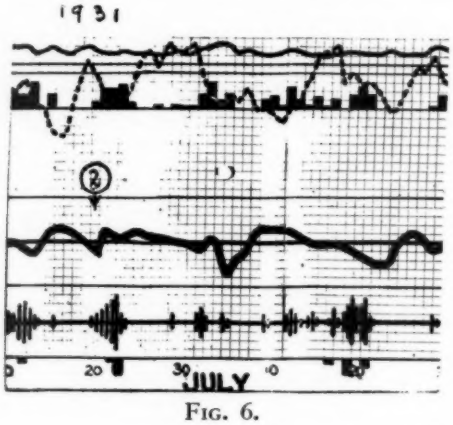

Fig. 6.

death was caused by the sudden infall of polar air. ⑮ occurs clearly with the onset of a polar episode.

Four deaths occur in the autumn and these are characteristically grouped at one particular period of the year. Of these 4 deaths, ⑯ and ⑰ occurred with the onset of a great polar episode at the crest of the barometric pressure with low temperatures on November 22.

The next 2 cases ⑱ and ⑲ occur simultaneously with the onset of the next great barometric crest. It must be apparent that the relationships of the episodes to each other and to the environment are so characteristic that further comment hardly

seems necessary. With the exception of case ⑭ we can definitely determine that every case of death occurred at a time when

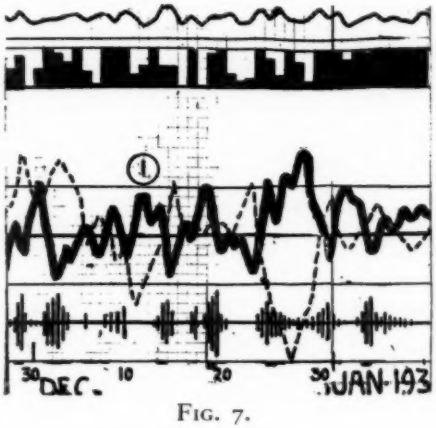

Fig. 7.

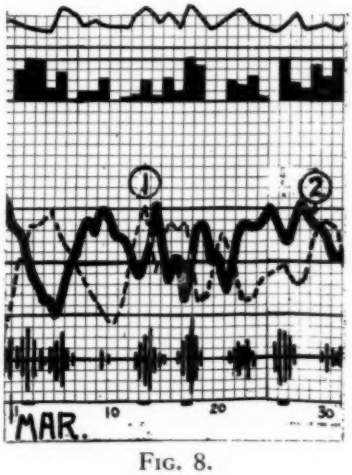

Fig. 8.

the environment demanded great adjustment on the part of the individual.

high wind, etc., with a sharp turndown in barometric pressure but with relatively lower temperatures. It probably reflects

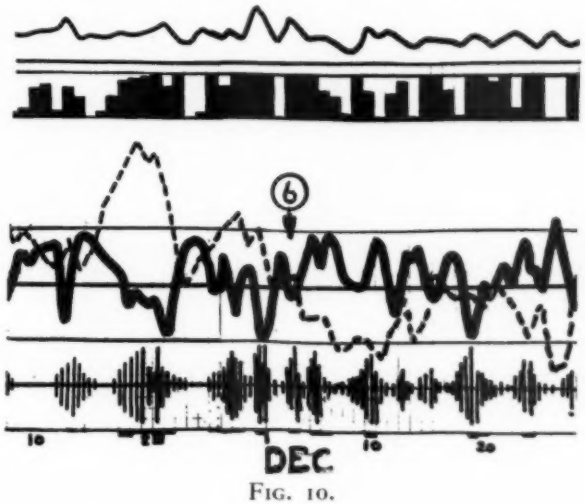

Fig. 10.

an occluded cyclone.

② occurs at the time of a polar infall with the event indicated by falling temperatures and sudden rise in barometric pressure.

1933

The only case examined occurred at the crest of a polar episode on December 12.

1934

During 1934 6 cases were examined. Their meteorological associations are indicated in the following meteorographs.

① March 13. This occurs at the time of a

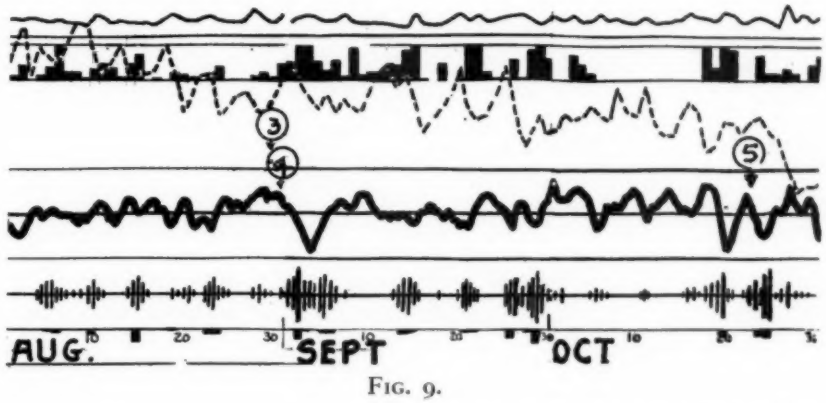

Fig. 9.

1931

During this year 2 cases were examined;

① occurs on March 7. This date corresponds to a time of an unusual storm, rain, polar infall, the barometer beginning to rise after the evening of the 12 and reaching a crest on the 14. The environmental temperature was still relatively high on the 13.

② March 29. Death occurs on a secondary polar infall after the barometric crest of the 28.

death of "status lymphaticus" finds its logical explanation in death due to a sudden chemical and endocrine upset of the type

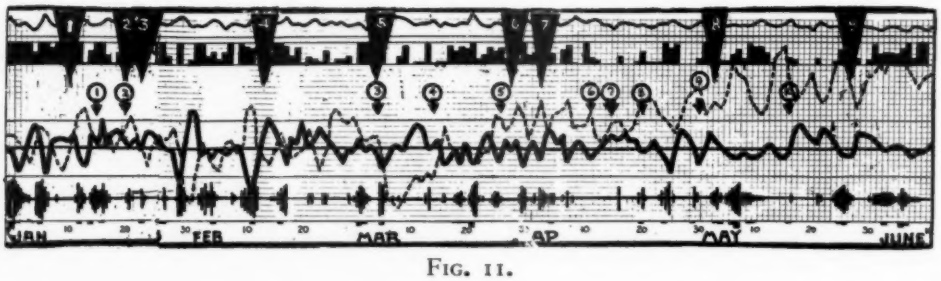

FIG. 11.

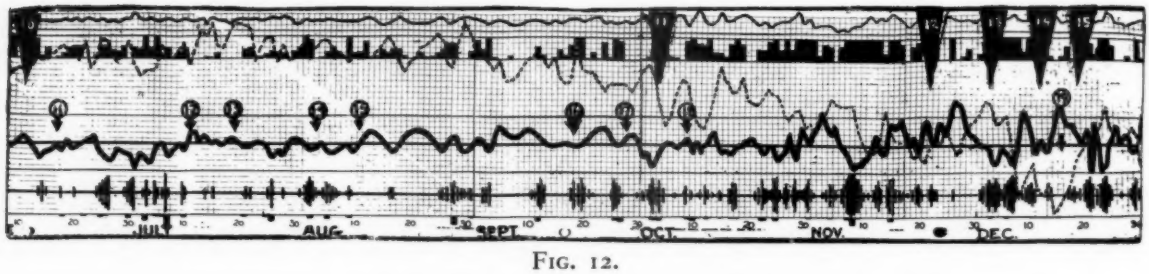

FIG. 12.

FIG. 13.

FIGS. 11, 12 and 13. Circled arrows indicate thymic deaths; triangles indicate deaths from Addison's disease.

Cases ③ and ④ occur simultaneously at the end of a series of minor polar infalls. It will be observed that the barometric pressure was at a crest.

⑤ occurs with a barometric crest on the October 23.

The final case ⑥ occurred during the course of a series of polar infalls immediately in the wake of a sharp barometric crest which reached its maximum on the evening of the December 2. The subject died on the December 3.

In view of the meteorological associations here determined, the emphasis that Paltauf placed on the fact that an anatomical change is not necessarily involved in sudden death of "status lymphaticus" finds its logical explanation in death due to a sudden chemical and endocrine upset of the type that Petersen has determined as common with meterological changes.

I have already mentioned that no cases were examined during 1932. However, I have proceeded to examine the association of deaths from "status lymphaticus" for the year in a different fashion.

1932

By courtesy of Dr. Herman Bundesen, President of the Chicago Board of Health, I have tabulated the 19 cases for the year reported on the death certificates as dying from "status lymphaticus" and we can analyze the meteorological association by

indicating the episodes on the meterology for the year.

It is an episode such as that numbered ⑲ that makes the meteorological associa-

Episodes Numbered on Graph (Circled Arrows Numbered)	Date	Number of Deaths*	Meteorological Status
1	Jan 15	1	Polar.
2	20	1	A tropical front; barometer declining.
3	Mar. 4	1	A tropical front but during the course of the development of a severe cold wave.
4	14	1	Polar infall.
5	26	1	Polar front begins.
6	Apr. 11	1	Polar front.
7	14–15	2	Polar front; barometric crest.
8	20	1	Tropical; precipitation.
9	30	1	Polar front.
10	May 16	1	Unusually sharp polar infall.
11	June 17	1	Polar crest.
12	July 11	1	Polar crest.
13	19	1	Polar; barometric crest.
14	Aug. 3	1	Polar infall.
15	12	1	Polar infall.
16	Sept. 18–19	2	End of polar episode with sudden tropical front.
17	28	1	Polar front.
18	Oct. 9	1	Polar front.
19	Dec. 16	1	Polar maximum.

* Material obtained from the Chicago Health Department.

tion most plausible, when in the last one hundred days of the year it is the crest of the final cold wave which precipitates the death of the type that we label as "status lymphaticus."

While in this series of cases we cannot be certain that the diagnosis has been confirmed by autopsy, the meteorological associations again clearly indicate that the fatal episodes have almost always occurred at a time when severe meteorological strain involving marked chemical and endocrine adjustment must have occurred.

If this surmise is true then it might seem logical that individuals ill from Addison's disease might die at like periods; perhaps even on the same dates when death occurs from "status lymphaticus."

We turn back once more to the curve for the year 1932 and on it, it will be noted that I have indicated deaths from Addison's disease by triangles extending down and supplied by a number (1–15). These deaths occurred from January 10 to December 20. Now we note that this surmise seems to be born out by the facts.

DEATHS FROM ADDISON'S DISEASE DURING 1932*

Case 1	January 10	Occurs with a polar episode.
Case 2	January 20	Occurs with a polar episode on the same date as thymic death ②.
Case 3	January 23	Occurs with a polar episode.
Case 4	February 13	Occurs with a major barometric episode.
Case 5	March 4	Occurs with a major polar episode and death occurs at the same time as thymic death ③.
Case 6	March 28	The same meteorological episode that involves thymic death ⑤.
Case 7	April 3	A tropical crest, beginning of a polar infall.
Case 8	May 3	Polar episode, the same one that causes death for thymic case ⑨.
Case 9	May 27	A sharp polar episode.
Case 10	June 11	A polar crest.
Case 11	October 4	A polar crest, beginning of the same episode that terminated thymic case ⑱.
Case 12	November 21	Polar crest.
Case 13	December 3	Polar infall.
Case 14	December 13	Polar infall. Precedes thymic death ⑲ which occurred on the succeeding major barometric crest.

* This material obtained from The Chicago Health Department.

Insofar as such statistical material is unreliable, it will support the theory that the death in Addison's disease also occurs when adrenal inadequacy would be manifest most acutely. The fact that in ⅓ of the cases in Addison's disease the death occurs with the same meteorological episode that causes death in "status lymphaticus," in 2 instances with identical days, would but strengthen the general thesis.

CONCLUSIONS

1. In view of the absence of any macroscopic and microscopic findings to account for unexpected death, and in the presence of an enlarged thymus and spleen, hyperplasia of the lymph glands with prominent Peyer's patches, associated with hemor-

rhage in Peyer's patches and intestinal tract, the diagnosis of "status thymicolymphaticus" was made. This diagnosis I have employed in the unexpected deaths, particularly in infants and children, when these cases were referred to the Coroner's office, and when an autopsy presented evidence indicating thymic death.

2. Pathologic-anatomic proof for death can not always be demonstrated. The absolute proof of the cause of death might be a functional disturbance associated with altered biochemical changes, disturbed pathological-physiological alteration, or constitutional variability markedly disturbed by extrinsic agents such as physical and meteorological climaxes.

Status thymicolymphaticus is a constitutional type. Sudden death here might occur when an unusual meteorological situation might require prompt physiological adjustment.

3. In view of the meteorological associations here determined, the emphasis that Paltauf placed on the fact that an anatomical change is not necessarily involved in sudden death of "status lymphaticus" finds its logical explanation in death due to a sudden chemical and endocrine upset of the type that Petersen has determined as common with meteorological changes.

4. In a period of four years there were 28 deaths in infants and children on whom an autopsy was performed and a diagnosis of status thymicolymphaticus was made. These autopsies were performed on deaths referred to the Coroner's office as "sudden" or "unexpected deaths." In one year, 1932, the death certificates from the Chicago Health Department were investigated and indicated 19 deaths from "status lymphaticus."

5. In order to correlate the association of the adrenal with the thymus gland in reference to "sudden death," 15 cases of Addison's disease are compared with thymic deaths for the year 1932 and the series of deaths have been charted on meteorographs with the dates when deaths occurred.

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DIFFERENTIAL DIAGNOSIS OF HYPERTHYROIDISM*

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THE diagnosis of Grave's disease in its classical form can be made almost at a glance, but unfortunately hyperthyroidism does not always appear in such a startling manner. The toxic manifestations of the so-called secondary type of hyperthyroidism are frequently dominated by only one of the component symptoms. The heart symptoms so often overshadow the clinical picture that they comprise one of the most difficult problems in the diagnosis of thyroid toxicity. This so-called secondary form of hyperthyroidism is that which occurs in a preexisting goiter, usually an adenoma although it may be in an old colloid hypertrophy. The histological changes in the gland begin in young adult life but evidence of toxicity is delayed until middle life when symptoms appear insidiously. This deceptive onset, together with the predominant cardiac manifestations, causes treatment to be given for the heart alone with resultant delay in the cure of the underlying disease.

In cardiac cases that fail to respond adequately to sound medical management, hyperthyroidism should be suspected. Digitalis alone will not influence materially the tachycardia and palpitation so common to hearts affected by thyroid toxicity. The patient may seek relief when rapidity of the rate or rapidity and irregularity of the beat are the only symptoms, but sometimes help is not sought until decompensation is present. Persistent tachycardia without obvious cause and especially when associated with auricular fibrillation, demands that hyperthyroidism be ruled out.

The clinical history plays an extremely important part in the recognition of "masked hyperthyroidism."[1] Careful questioning is necessary usually to reveal the symptoms hidden by the pronounced heart complaints. The patient is found to be aware of an increased nervousness with emotional instability, the body warmth is noticeably increased, frequently with hyperhidrosis and hot weather is tolerated poorly. Fatigueability and weakness are universal complaints. The food intake is as good or better than the previous average and yet is accompanied by a loss of weight. These two factors are very reliable findings because with increased metabolism, body tissue must be utilized with consequent weight loss, or the food intake must be increased to meet the greater demand. While it is possible for the food intake to be sufficient to prevent loss of weight, it is usually a combination of the two that is found. The association of a weight loss with a large food intake is seen only in hyperthyroidism and diabetes mellitus;[2] the latter disease being easy to identify by proper blood and urine studies.

Physical findings are not so striking as those seen in exophthalmic goiter. Lid-lag, lack of convergence and absence of forehead wrinkling on looking up are common but exophthalmos is rare. The skin is warm and moist; the outstretched fingers present a fine tremor. Except for the rapid rate and the pulse deficit, if present, the heart shows little change. Slight hypertrophy may be detected but marked enlargement and organic murmurs are absent in the heart of hyperthyroidism. The tachycardia is persistent, even during bed rest. Careful palpation of the neck reveals the presence and character of the thyroid pathology.

The following illustrative case reveals how these diagnostic historical and physical findings may be misinterpreted:

* Read before the Albany County Medical Society, October 23, 1935.

Case 1. Miss L., aged fifty-six years, was admitted to the Albany Hospital on September 25, 1935. Her present illness followed the physical and emotional strain of caring for a member of her family during a long cardiac illness terminated by sudden death.

In January, 1935, she experienced an attack of substernal oppression, associated with orthopnea, which she described as "breathing in a thick fog." This distress lasted but a few hours and has recurred on two occasions. From the onset tachycardia and palpitation have been present, made worse by exertion and more noticeable on lying down. Irregularity of the heart beat was noted. There was no other chest pain and no symptoms to suggest decompensation. On more careful questioning she admits increasing nervousness, irritability and emotional instability since December, 1934. Weakness of increasing severity has been present. There was no loss of weight but the appetite had been exceptionally good and the food intake large in amount.

A diagnosis of heart disease was made in January and digitalis was given. Although attention was directed to a goiter which had been present for eight years, medical advice in two instances assured her it was not responsible for her illness. The third medical opinion considered hyperthyroidism as the etiological agent of her complaints and prescribed iodine with benefit to the patient, who then delayed further treatment until coming to Albany this fall.

On examination the thyroid was found to be enlarged by bilateral, hen's egg-sized adenomata. The heart was not enlarged but a rapid rate with a total irregularity of the pulse was present. The apex rate was 140 and that at the wrist 120. The blood pressure was 150/90. The skin was warm and moist and there was a fine tremor to the outstretched fingers. The balance of the physical examination was without bearing on the present illness and laboratory studies of the blood and urine revealed no unusual pathology.

Digitalis and Lugol's solution were given and when the maximum benefit had been obtained, a subtotal thyroidectomy was done. The thyroid gland was found to be almost entirely replaced by multiple adenomata. The pathologist reported multiple adenomata of the thyroid gland which showed areas of hemorrhage and edema. From the sections studied a histological diagnosis of overactivity could not be made.

The patient, however, showed very definitely that toxicity had been present, for postoperatively the pulse, after the immediate rise, fell to normal levels. Auricular fibrillation was still present but so markedly reduced in severity that on the nurses record the apical and radial rates were charted on many occasions as being the same. Subjectively the patient noted complete relief from her symptoms.

In the cardiac group, then, the problem is primarily one of accurate recognition which is dependent upon a careful history and physical examination for its successful solution.

The neuroses present a problem in which the difficulties encountered are more truly those of differentiation. Although many forms of neurosis may mislead the examiner, it is more commonly that group of young adults suffering from cardiovascular neurosis. This condition, also known as disordered action of the heart, neurocirculatory asthenia, effort syndrome and soldier's heart, may imitate hyperthyroidism with great fidelity.

Cardiovascular neurosis occurs in persons having a constitutional weakness that tends to be familial. The onset of symptoms of this entity is vague as contrasted with the rather definite beginning of hyperthyroidism. Symptoms for which no organic change can be demonstrated are common. These patients are usually mentally depressed, have little ambition but may still accomplish a great deal of work. Mayfield[3] aptly expresses this state as one of chronic fatigue. Hyperthyroidism, on the other hand, predisposes to optimism; work is begun with enthusiasm but a genuine weakness soon causes the individual to stop. With cardiac neurosis the patient may or may not show weight change but the appetite is almost always poor. Tachycardia is common to both, but in the patient with the neurosis the heart rate is extremely variable. An important point in the diagnosis of this condition is a drop in the pulse rate from 120 or more, to normal following a few hours of bed rest.

Objectively and subjectively, cardiac neurosis presents cold, moist hands and

feet. Hurxthal[4] feels that the presence of this finding is sufficient evidence to almost immediately exclude hyperthyroidism. Likewise, this writer always doubts the existence of hyperthyroidism in a patient complaining of cold hands and feet, especially when acrocyanosis and mottling are also present. When noted, the tremor of the outstretched fingers in cardiac neurosis is coarse. Typical eye signs of goiter are absent from the findings of this disease.

When subjected to the determination of basal metabolism, a neurocirculatory asthenic will usually show a high metabolic level on the first test. This unfortunate fact has been responsible for much unnecessary surgery. Repeated observations are imperative if the actual basal metabolic level is to be determined.

Case II. Mr. S., aged twenty-three years, who was admitted to the Albany Hospital on Sept. 9, 1934, is representative of the cardiovascular neurosis group. He stated that for the past three months he had experienced attacks of tachycardia and palpitation. Slight dyspnea was noted on exertion. During the present illness increasing weakness and fatigue had forced him to stop work. A tremor had been present from childhood but during the past six months had increased greatly in severity. The appetite had been poor but no significant weight change occurred. A metabolic rate taken in another city was reported as plus 34 per cent and because of this thyroidectomy had been advised.

Examination failed to reveal any eye signs of goiter. The thyroid gland was entirely normal to palpation. The heart was of normal size and position, the rhythm regular and the rate 100. There was a coarse, irregular tremor of the hands, which were cold and moist. The balance of the clinical and laboratory examination was negative including the Wassermann report. Metabolism determinations on two successive mornings were plus 18.4 per cent and plus 5.9 per cent.

The patient was discharged without operation and has improved with the general hygienic measures employed to combat his disease.

The importance of the determination of the metabolic rate has been greatly overemphasized in the diagnosis of hyperthyroidism. As just shown in the last case, an unfortunate surgical experience might result from too great dependence on a test that, at most, is only one point to be considered in the diagnosis. The true basal metabolic rate is seldom obtained by only one determination, and a metabolic rate that declines to normal levels on successive days almost entirely rules out hyperthyroidism.[5] No test, least of all one in which the opportunity for error is so great, can supplant careful evaluation of the clinical history and findings. It is the opinion of the writer that fewer mistakes will be made and much less needless surgery performed if the diagnosis of hyperthyroidism be made entirely without the aid of a metabolic rate determination.

Other instances of the fallacy of dependence on the metabolism test for diagnosis, are found in the group of non-toxic nodular goiters whose obvious size and pathology make operation necessary and yet, when the metabolic rate is determined it may be as low as minus 20 per cent. Still another group have actual hyperthyroidism with a normal metabolic rate.[6] In such a patient reliance must be upon clinical judgement in order to determine the course of action. This is well illustrated in the case of:

Case III. Mrs. B., aged forty-five years, who was admitted to the Albany Hospital on September 24, 1933. The history revealed frontal headaches of daily occurrence and increasing prominence of the eyes for two months; choking sensations in the neck and dyspnea for six months; nervousness and restlessness for one year. A good appetite had prevented weight loss.

On examination hyperhidrosis was noted. Exophthalmos, though moderate, was obvious and with it the other classical eye signs of goiter were noted in mild degree. Satisfactory palpation of the thyroid gland was impossible due to a thick, short neck. Her blood pressure was 118/78. The pulse rate was 110. Metabolism tests were as follows: on September 25, minus 12.5 per cent; on September 26, minus 12 per cent; and on September 30, minus 13 per cent. The pulse rate declined to 90 under the usual

preoperative treatment of rest and iodine. A subtotal thyroidectomy, done on October 2, showed the gland to be four times normal size, vascular and friable in character beefy in appearance with evidence of excess colloid. Microscopical examination of the tissue removed showed mild hyperplastic changes in an old colloid goiter.

Postoperatively, auricular fibrillation was present for five days but disappeared and a normal rhythm with a rate of 90 existed at the time of discharge. Subjectively the patient was relieved of her symptoms. She was last seen on April 22, 1935, at which time the pulse rate was 80; there had been a gain of fifteen pounds in weight and the patient was clinically well.

If the metabolic rate had been depended upon for diagnosis in this case, as too often it is, this patient would have been denied good health.

SUMMARY

1. The recognition of hyperthyroidism when the cardiac symptoms dominate, is dependent upon a carefully taken history and thorough physical examination. Failure to obtain results with the medical management of heart disease should arouse suspicion of thyroid toxicity.

2. Cardiovascular neurosis may present nearly all of the symptoms of hyperthyroidism. Differentiation of the two conditions may require prolonged study. Again, a complete history and painstaking examination of the patient are the most important diagnostic aids.

3. The true basal metabolic rate can often, only be determined by repeated observations. The test is of little practical value in the differential diagnosis of hyperthyroidism. Neither the diagnosis nor the therapy of goiter can be determined by a test in which the opportunity for error is so great.

I am indebted to Dr. George E. Beilby for permission to present his cases from the Albany Hospital.

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APPARATUS FOR TREATMENT OF FRACTURED OS CALCIS*

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SINCE the incidence of fracture of the os calcis is small when compared to those of other bones, the percentage requiring radical surgical intervention is proportionate. This observation is based on the fact that the majority of fractures of the os calcis are simple fissure fractures without much displacement. However, when there is a badly displaced simple or comminuted fracture of the os calcis it becomes a serious, social, economic and compensation problem.

Stimson[1] in a review of the literature on fractures for 1933 and 1934, summarizes the status of the management of fractures of the os calcis.

Fractures of the os calcis present an extremely interesting problem. They have a notoriously poor prognosis because of the persistent pain on weight-bearing, and various means have been devised in an attempt to improve the results. There are two main schools. One believes in the closed method advocated by Boehler in which wires are inserted in the posterior part of the os calcis and in the tibia with a force pulling them apart to correct the deformity (Schinder, Forrester, Stewart, Hope Carlton). The other favors immediate open reduction, recommending for fractures with downward crushing of only the articular surface a simple elevation of the surface with chip-graft supports (Lenormant, Sorrel), and for complete fractures through the body with upward angulation a wide exposure with reduction and osteosynthesis of some form with or without tenotomy of the Achilles tendon (Wertheimer, Secor and Mutricy, Gregoire and Couvelaire, Denny, Leriche). It is difficult to evaluate the two methods as each is advocated by men of great experience.

[1] STIMSON, BARBARA B. A Review of the 1933–1934 Literature of Fractures. *Surg. Gynec. and Obst.*, 62 (Jan.) 1936.

The purpose of this paper is to describe a simple apparatus for reduction of fracture of the os calcis and to illustrate its efficacy in 6 cases of badly comminuted and displaced fractures. No attempt is being made to review the literature or to make comparisons with other forms of treatment.

This is a preliminary report; the final evaluation of treatment by the apparatus must come after several years of use by those interested in this problem.

APPARATUS

Basically, the apparatus is a simple device for reducing fracture of the os calcis and maintaining the reduction and consists of a universal fitting central bar, Fig. 1B which fits in the concavity formed by a plaster boot applied with the foot at right angle and with internal rotation. The straps, A and G allow fixation of the central bar to the plaster boot at the foot and calf regions. The brace, F, is affixed to the central bar, B. This member strengthens the central bar and forms the support for the traction screw, C. The stirrup, J, has its fulcrum at N and allows the motion of the stirrup through arcs L and M. The screw, C, operates through the swivel joint, D, which is attached to the central bar of the stirrup, so that rotation of the screw, C, allows anterior and posterior motion of the stirrup with great force through arcs L and M for a distance of 10 cm. The arms of the stirrups, J, are constructed so that they can be elongated for 5 cm. by rotation of the screws, E. At the ends of the stirrup arms, J is an adjustable clamp, P, which grasps and fixes the Steinman pin to the arms of the stirrup.

* From New York University College of Medicine and 3rd Surgical Division Bellevue Hospital, Arthur M. Wright, M.D., Director.

Figure 1A is a side view of the apparatus as it is set for application to the foot over the plaster boot. The stirrup is rotated as far backward or upward as possible, thus placing it at right angles to the concavity of the central bar. The stirrup arms, J are shortened through operation of the screws, E, enough to allow the stirrup arms to grasp the ends of the Steinman pin by clamps, P. Figure 2A is a front view of the apparatus with the stirrup arms fully lengthened and the stirrups anteriorly rotated to the fullest extent. Figure 2B shows the apparatus as it appears when applied to a patient.

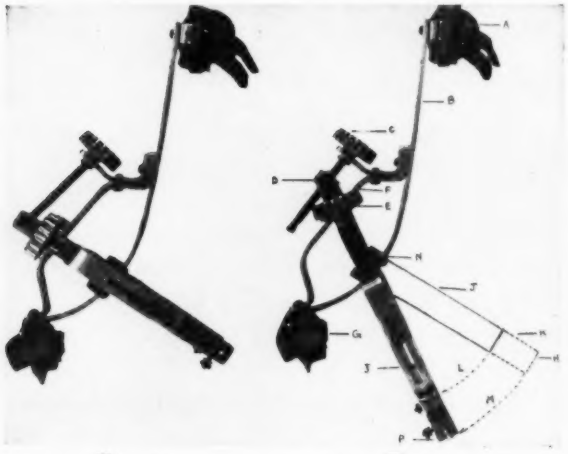

FIG. 1A. FIG. 1B.

FIG. 1A. Apparatus set for application to the patient. Traction stirrup (J) rotated posteriorly and shortened as much as possible.

FIG. 1B. (A and G) Straps for fixing apparatus to leg, ankle and foot over the cast. (B) Universal fitting curved bar. (C) Screw fixed to apex of stirrup at swivel joint (D) rotates stirrup (J) from position H to J. (E) Screws affixed on jointed stirrups (J) which lengthen and shorten them. (F) Brace for curved bar (B) and support for screw (C). (N) Fulcrum for rotation of traction stirrup. (L) Range of motion of stirrup when shortened. (M) Range of motion of stirrups when lengthned. (K) Range of lengthening of stirrups (J). (P) Adjustable clamps for fixing stirrup to Steinman Pin.

APPLICATION OF APPARATUS

Under spinal anesthesia and sterile precautions, a Steinman pin, six inches long and which can be unscrewed in the center, is passed through the os calcis at a point 1 to 2 cm. below the attachment of the tendo-achilles.

In the first 3 cases to be described, tenotomy of the tendo-achilles was not performed, but was done in the second group of 3 cases. While not absolutely essential, tenotomy is highly recommended as it prevents any slight displacement caused by the pull of the tendo-achilles when the apparatus is removed.

FIG. 2A.

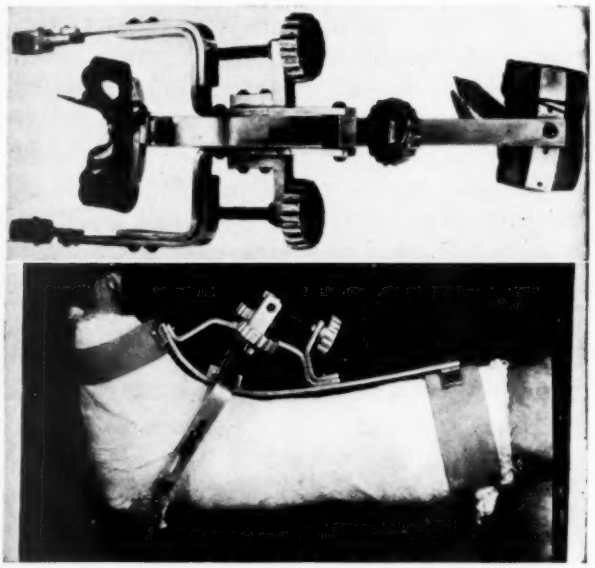

FIG. 2B.

FIG. 2A. Front view of apparatus with stirrups fully lengthened and fully rotated forward.

FIG. 2B. Apparatus applied for reduction of fracture of os calcis.

Sterile dressings are applied to the points of entrance and exit of the Steinman pin and to the operative wound through which the tenotomy was performed.

A plaster boot is then applied to the affected side including the foot and leg below the knee joint, with the foot at right angles and adduction but allowing the heel to remain outside the cast. This fixation of the foot allows any traction applied to the os calcis to act on that bone without movement of any other part of the foot or ankle. When the plaster boot is thoroughly hardened and dried, the apparatus is strapped onto the foot and leg as shown in Figure 2B, the adjustable clamps at the end of the stirrup grasping the Steinman pin on either side. The points of the pin

are covered with corks for protection to the patient, nurses, tearing of bed linen, etc.

occurred to either side, simple overlengthening of one of the stirrup arms will correct this deformity. An *x*-ray plate is

Fig. 3A. Case 1. H. H. Fracture right os calcis before reduction.

Fig. 3B. *X*-ray film of reduction obtained after removal of apparatus.

The screws, E, are next operated to lengthen the stirrup arms backward. The lengthening is continued until all impaction is broken and the widening of the body of the os calcis is properly reduced to normal. The screw, C is then operated to rotate the

taken immediately and further adjustments are made until the fragments are in perfect alinement.

The apparatus is made of duraluminum and weighs less than one pound. The force through which the screws are operated is

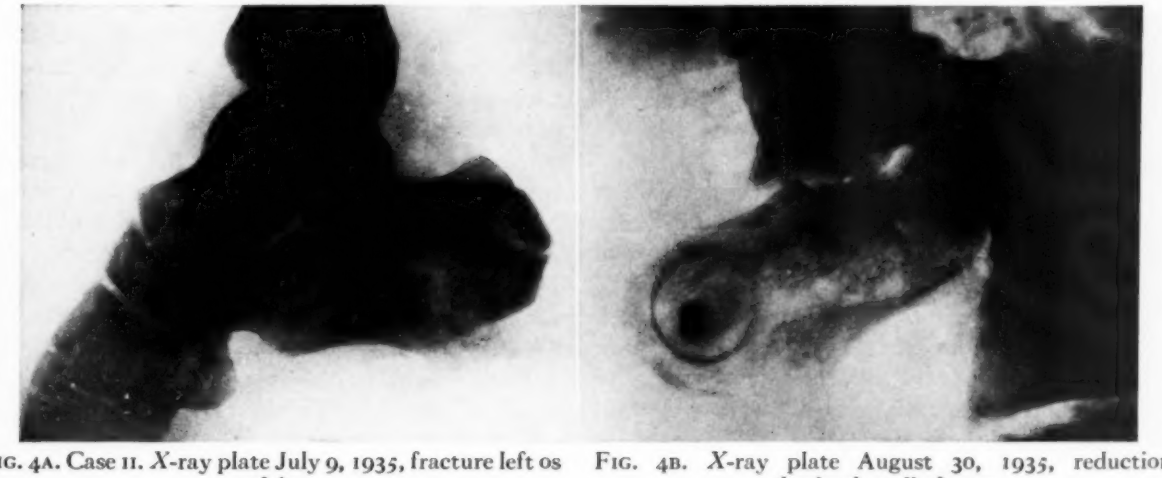

Fig. 4A. Case II. *X*-ray plate July 9, 1935, fracture left os calcis.

Fig. 4B. *X*-ray plate August 30, 1935, reduction obtained at discharge.

stirrup arms through arc L and M. (Fig. 1B) which rotates the fragments downward and forward. This represents the exact opposite force which caused the fracture, namely, upward and backward. In the event that tilting of the fragments has

tremendous and readily reduces the fracture. The apparatus can be removed immediately after reduction and the plaster boot completed to incorporate the Steinman pin in the exact position of reduction by the apparatus. The plaster surrounding the pin

holds the proper alinement of the fragments. This can readily be followed when previous tenotomy of the tendo-achilles has been performed. In two to six weeks the pin is removed by unscrewing it at its center and each portion pulled out to the right and left, not endangering the surgical sterility of the pin wound.

The apparatus may be allowed to remain in place one, two or three weeks and then removed and the plaster boot completed over the pin. When *x*-ray pictures reveal the proper holding of the fragments by the plaster boot, the pin is removed as described. This is the procedure used in the following cases. Immobilization is maintained for eight weeks.

Fig. 5A. Fracture of the left os calcis before reduction.

Fig. 5B. Result obtained on discharge of patient.

CASE REPORTS

Case I. H. H. was admitted to Bellevue Hospital on June 16, 1935, with a history of having fallen from one roof to another at a height of thirty feet. He had extreme pain in his left thigh just above the knee, with deformity of the leg and was unable to arise and bear weight. The patient also suffered severe pain in his right foot just below the ankle with some swelling of the heel.

On physical examination there is deformity at the lower third of the left thigh with widening of the area just above the knee. The patient can move his hip but not his knee. Palpation reveals two fragments, crepitation, overriding of the upper fragment, false point of motion and swelling in the lower third of the femur. There is a small amount of swelling and ecchymosis of the heel and ankle of the right lower extremity with no gross deformity with crepitus and extreme tenderness in the os calcis. The rest of the physical examination is negative. Laboratory data are all negative. *X*-ray film showed a comminuted fracture of the right os calcis and the lower extremity of the left femur with backward and outward displacement of the lateral condyle.

Course. On July 19, 1935, under spinal anesthesia, a Steinman pin was inserted through the right os calcis, 1 cm. below the attachment of the tendo-achilles. Sterile dressings were applied to the points of exit and entrance of the Steinman pin and a plaster boot applied to the foot, ankle and leg, allowing the entire heel to remain outside. The apparatus was strapped on the leg over the plaster boot, as shown in Figure 2B, and reduction was performed as previously described.

August 25, the plaster boot and pin were removed and physiotherapy instituted. By the time of discharge, September 10, the patient was able to use the leg and foot without pain and could bear weight with great comfort.

Figure 3A illustrates the *x*-ray appearance of the fracture before reduction, and Figure 3B the result obtained on discharge of the patient.

Cases II and III. A. G. was admitted to Bellevue Hospital on July 9, 1935, with a past history of long alcoholism and many previous injuries. On July 2, he had received a fracture of the jaw. The patient states he was being pursued by several individuals (hallucination?) and jumped two stories landing on both feet

and was unable to arise. He was acutely intoxicated at the time.

On admission, a physical examination revealed an acutely intoxicated, well nourished, twenty-seven year old white male. Positive findings were the bilateral fracture of the os calcis and fracture of the jaw.

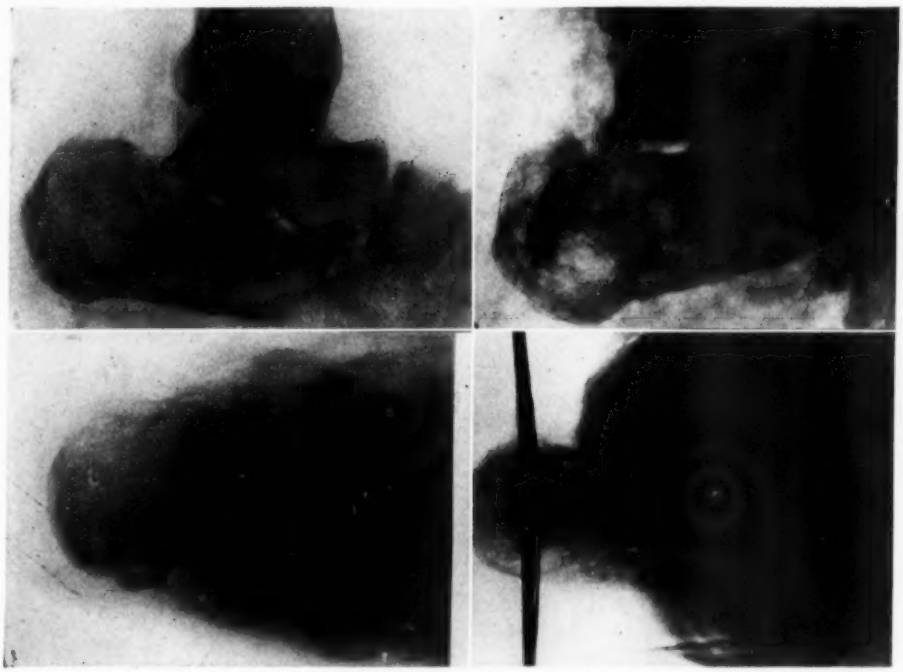

Fig. 6. Upper left to right: (1) Fracture of os calcis before reduction. (2) Result obtained on discharge of patient. Lower left to right: (1) Impaction before reduction. (2) Reduction of impaction with pin in place.

On July 11, under spinal anesthesia, a Steinman pin was put through the left os calcis, 2 cm. below the insertion of the tendo-achilles; a plaster boot was applied, and the Reduction Apparatus was strapped in place. Postreduction *x*-ray pictures showed excellent reduction. The same night, during an hallucinatory episode, the patient removed the appliance. It was reapplied, re-*x*-rayed and again good position was reported.

On July 18, the apparatus was removed from the left heel. An *x*-ray film showed no change in position. The plaster boot was kept on, the heels being covered with plaster after removing the Steinman pin.

On July 15, the apparatus was placed on the right heel and removed on August 8. This time the Steinman pin was left in the heel for traction and the area was covered with plaster. On August 16 the pin was removed.

On August 20, the circular casts were removed from both legs and posterior moulded splints were applied. On August 28, 51 days after the accident, the posterior moulded splints were removed and on August 31 the patient was allowed up on crutches.

During his hospital stay, the patient complained early of much pain in both heels and feet; towards the latter part of his course, only of heel pain at night. On September 4, he was up and about on crutches and could place weight on both heels with only slight local pain. He is being treated in the Physiotherapy Department and promises to return regularly after being discharged.

Figure 4A shows *x*-ray picture of the fracture of the left os calcis before reduction, and Figure 4B the reduction obtained on discharge of the patient.

Case IV. S. L., aged fifty-four years, gave a history of stepping off a curbstone, twisting his left foot and sustaining an injury to his left os calcis. *X*-ray picture revealed a comminuted fracture of the posterior segment of the left os calcis with some upward displacement of the upper fragments. Under spinal anesthesia, October 30, the left tendo-achilles was divided and a Steinman pin introduced according to the foregoing description. A plaster boot was applied and the apparatus strapped on and reduction affected. All laboratory data, pre- and postoperatively, were negative. Postreduction *x*-ray films showed

approximation of the fragments and correction of the widening in the anteroposterior view. The apparatus was removed November 10, apparatus applied over a plaster boot. Postoperative check-up plates revealed a satisfactory position of the fragments with proper

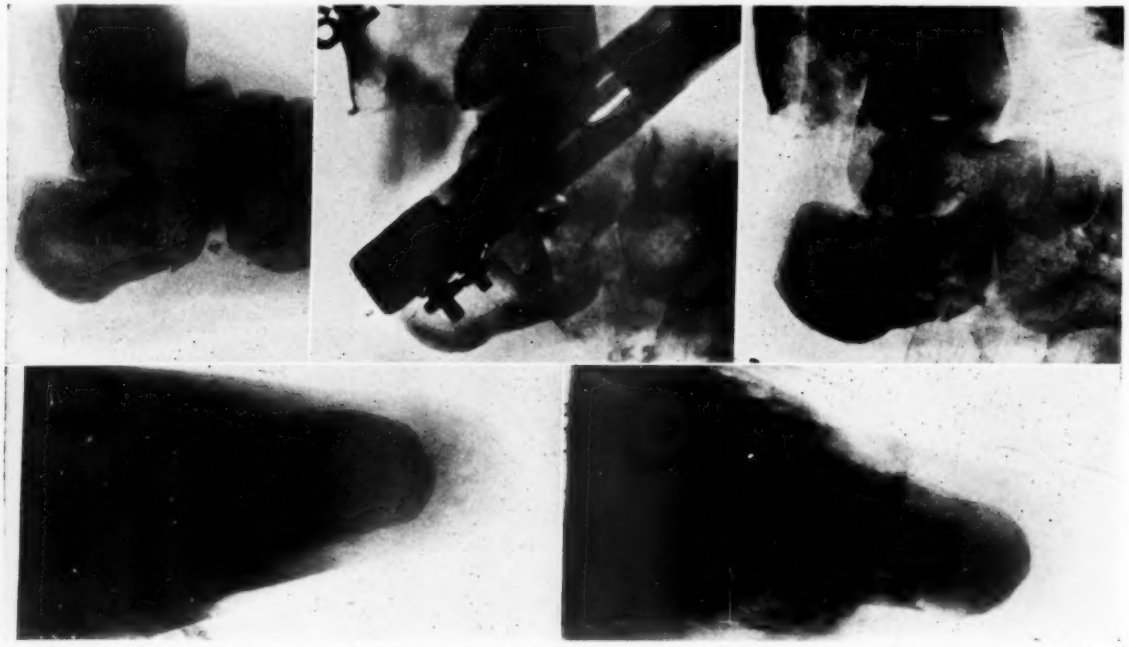

Fig. 7. Upper left to right: Fracture before reduction; fracture reduced and apparatus in situ; result obtained on discharge of patient, 50 per cent loss of reduction, patient removing pin and apparatus ten days postreduction. Lower left to right: impaction of body of os calcis before reduction; impaction corrected.

the Steinman pin being left in place and the plaster boot completed over the pin and heel. The pin was withdrawn November 18, and final plates showed the os calcis fragments to be in good position.

Figure 5A represents *x*-ray picture before the reduction, and Figure 5B, result obtained on discharge, eight weeks after application of apparatus. On January 15, 1936, the patient reported to the Return Clinic and had no complaints.

Case v. J. B. aged forty-nine years, was admitted to Harlem Hospital after a fall from a first story window. He sustained a Pott's fracture in left ankle and a fracture of the os calcis in the right foot. The patient was transferred to Bellevue Hospital on November 3, 1935. As the patient was psychopathic and unmanageable, an attempt at reduction was not made until November 13. Prereduction *x*-ray plate showed a comminuted fracture of the right os calcis with upward displacement of the posterior fragment. All laboratory data were negative otherwise. On November 13, under spinal anesthesia, tenotomy of the tendo-achilles was performed, a Steinman pin introduced in the usual way and the reduction reduction of the lateral impaction. On November 26, the apparatus was removed and a boot completed around the Steinman pin and heel. On December 3, the Steinman pin was removed and *x*-ray plates showed a proper maittenance of the postreduction position. The cast was removed January 15, 1936 and physiotherapy instituted. While the patient could bear weight upon the removal of the cast, the final evaluation of the case cannot be made for several months.

Figure 6. Upper left to right; (1) Fracture of os calcis before reduction: (2) Result obtained on discharge of patient.

Lower left to right: (1) Impaction before reduction. (2) Reduction of impaction with pin in place.

Case vi. S. S., aged fifty-five years, was admitted on December 14, 1935, after having jumped from a front story window. On admission there was clinical evidence of a fractured left os calcis but *x*-rays could not be taken until December 18, 1935, patient being unmanageably psychotic. *X*-ray plate (Fig. 7)

showed a comminuted fracture of the os calcis with complete loss of tarsal arch. On December 26, the apparatus was applied in the usual way combined with tenotomy of the tendo-achilles. Postreduction *x*-rays showed good alinement of the fragments. On January 6, 1936, ten days postreduction, the patient being psychotic, took the apparatus apart and withdrew the Steinman pin himself. As a result, our efforts were defeated in that we were forced to complete the boot with some flattening of the arch. The patient was ordered discharged on crutches, on January 9. Final *x*-ray plates showed good alinement of the fragments in the uteroposterior view with some flattening of the arch of the os calcis in the lateral view. This case emphasizes the extreme care necessary in the management of this condition. On February 8, 1936, a report from the State Hospital reveals that patient is bearing weight on the cast without symptoms.

Figure 7. Upper left to right: (1) Fracture before reduction. (2) Fracture reduced and apparatus in situ. (3) Result obtained on discharge of the patient.

Lower left to right: (1) Impaction of os calcis before reduction. (2) Impaction corrected.

CONCLUSION

1. An apparatus is described to reduce fractures of the os calcis.

2. Only the very severe and badly displaced fractures of the os calcis have been chosen to determine the efficacy of the apparatus.

3. In this preliminary report of 6 cases, the apparatus readily reduced the fractures and on discharge of the improved patients, the *x*-ray findings and clinical course were satisfactory.

4. The final evaluation of these cases, or the ultimate value of the apparatus must come from several years of use and observation.

ACUTE ARTERIAL OCCLUSIONS OF EXTREMITIES*

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NO one can discuss the sudden arterial occlusions of the extremities without referring to the fundamental monograph of Einar Key published in 1929.[1] A more recent excellent collective review has been given by Herman Pearse, Jr. in 1933, who brought the number of reported cases of peripheral embolism up to 296.[2] These collective statistics are of great importance as the individual surgeon has hardly ever seen or reported more than a few cases, not exceeding 15 in one series. There are several reasons for the infrequency with which the individual meets this serious complication.

In the first place, embolism into arteries of the extremities is not as frequent as compared with embolism in other organs. Thus Bull[3] found in 6140 autopsies 301 emboli in 189 patients (Table 1). From his careful work it appears that embolism into peripheral arteries is one of the least frequent, embolism into lungs, kidney, spleen and brain heading the list.

TABLE 1
PERCENTAGE OF EMBOLISM IN 6140 AUTOPSIES*

Site of Embolism	*No. of Cases*	*Per Cent*
Lungs	113	1.8
Kidney	74	1.2
Spleen	60	1
Brain	32	0.52
Extremities	15	0.24
Intestines	6	0.1
Liver	1	0.016

* Bull. *Beitr. z. Chir.* 125: 519, 1922.

There is another important lesson to be learned from the statistics of Einar Key, Pearse and others, namely, that the overwhelming majority of embolisms treated with or without embolectomy were reported from the Scandinavian countries, chiefly Sweden, due to the recognition by Swedish physicians of the great importance of early treatment. Perhaps the great distances and relative paucity of well trained surgical residents in some of our big cities contribute to the fact that peripheral arterial occlusion is so often ignored or simply accepted as the inevitable forerunner of gangrene and amputation.

Very instructive is the source of embolism, as reported by Pearse (Table 11). The

TABLE 11
THE SOURCE OF PERIPHERAL EMBOLI IN 296 CASES*

	Per Cent
Heart disease	69.2
Postoperative states	13
Infection and trauma	2.1
Arteriosclerosis	2.1
Aneurysm	1.8
Abortion and delivery	1.8
Miscellaneous	2.8
Phlebitis	0.3

* Pearse. *Ann. Surg.* 98: 17, 1933.

main source of emboli to the peripheral arteries is the left heart, less frequently the aorta; the pulmonary veins need hardly be considered. Mitral stenosis and myocardial thrombi are the most frequent heart lesions. A group of arterial thromboses occur after operations, infectious diseases, and in aneurysms from where they may easily be thrust farther into the periphery of the arterial tree. Thus a brachial embolism after thyroidectomy or a femoral embolism after an abdominal operation has its source in an arterial thrombus in the surgical field, which grows by apposition

* From the Department of Surgery, University of Illinois, College of Medicine. Read before the North Side Branch, Chicago Medical Society, Dec. 5, 1935.

to the bifurcation of the main vessel and from here is thrown into the periphery. Arteriosclerotic plaques of the aorta may also be the source of peripheral emboli.

The embolus almost invariably gets stuck at the major bifurcation of the peripheral arteries where the lumen of the vessel suddenly decreases. The following combined diagram (Fig. 1) has been prepared on the basis of the work of Pearse,[2] Petitpierre[4] and Heidrich,[5] calculated in percentages. The axillary and brachial arteries on the upper extremity, the aorta and common iliac on the trunk, and the femoral and popliteal arteries on the lower extremity are the most frequent sites of embolism. It is very instructive to compare the frequency of the site of embolism with the danger of gangrene from a simple ligature at the same site.[5] It is of course well realized that these latter figures cannot be compared with the results of an embolic obstruction because the latter endangers collateral circulation to a greater degree. Nevertheless, they serve as a rough indication of the danger of gangrene and may thus permit or contraindicate expectant treatment. For example, in the case of brachial embolism which occurs in approximately 12 per cent of peripheral arterial emboli, the danger of gangrene from simple occlusion is small, being only 3.13 per cent. Thus active surgical measures will not often be necessary, and if performed, the success may not always be due to the embolectomy. On the other hand an embolus of the common iliac or femoral artery is followed by such serious circulatory difficulties that an embolectomy should be considered much more readily. It may be of help to every surgeon considering embolectomy to have such a chart before him which, with other factors to be discussed presently, would aid him in frequently difficult decisions.

The diagnosis of peripheral arterial embolism has been said to be easy. The classic description is that of a sudden excruciating pain at the site of the embolus in a patient suffering from heart disease; simultaneously the affected extremity becomes paralyzed, cold and pale, the pulses disappear, and in a few hours the skin

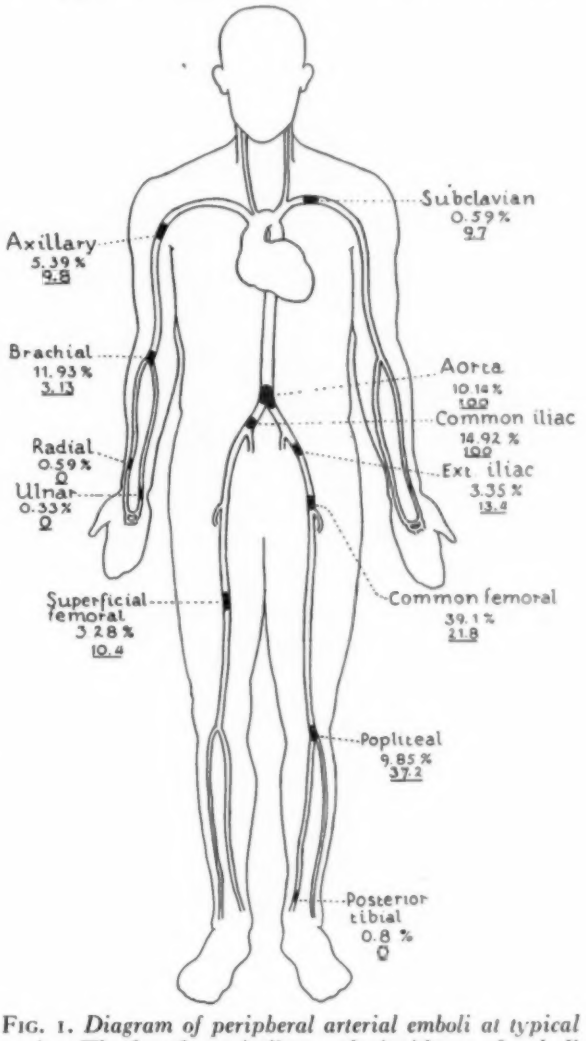

Fig. 1. *Diagram of peripheral arterial emboli at typical sites.* The first figure indicates the incidence of emboli in percentages calculated from the collective statistics of Pearse.[2] The underlined figures represent the percentage of gangrene following a ligature of the artery at the same site, based on the collective review of Heidrich.[5]

becomes mottled with a bluish hue, later showing definite cyanosis. On the fingers and toes, or sometimes over prominent bones such as the tibial margins, dark blisters appear which may open and from which the gangrene spreads, although I have seen a complete restoration of cutaneous circulation even at this stage. The pain of this acute ischemia is excruciating and often unrelieved by large doses of morphine.

As a contrast, arterial thrombosis comes on gradually. There is good evidence of preexisting peripheral arterial lesions such as arteriosclerosis or Buerger's disease. There is no obvious source of embolism in the heart or in a surgical field.

It has been my experience with 27 cases of sudden arterial thrombosis and 10 cases of arterial embolism, that the differential diagnosis between these two types of arterial occlusion is often clearcut, sometimes difficult, and occasionally impossible. It has been noticed by several observers that arterial embolism may have premonitory symptoms, that days or weeks before the sudden fulminating vascular occlusion, numbness, coldness or tingling may appear. These are preliminary showers of the central clot which is about to break loose. On the other hand arterial thrombosis has been observed in patients in whom such a sudden occlusion was the first indication of a widespread arterial disease. In 2 patients suffering from multiple segmental occlusions, a popliteal thrombosis was the first clinical symptom of Buerger's disease and could have readily been mistaken for embolism. Furthermore, diabetes and arteriosclerosis, which often lead to peripheral vascular disease with initial periods of acute thrombosis, may also lead to mural changes in the heart and to peripheral emboli from such a central source.

Acute vessel spasm following trauma, the so-called traumatic segmental vessel spasm, may be mistaken for an organic occlusion. The literature has been summarized and two instructive personal observations were reported by Montgomery and Ireland,[6] thus bringing the number of reported cases to 46. The authors explored the artery in both instances and found it free from clots but maximally contracted.

An acute vessel spasm accompanying a thrombophlebitis or lymphangitis may be mistaken for peripheral embolism. I have seen 2 cases, both of which were diagnosed as peripheral embolism and were found on careful examination to be deep venous thromboses. In the first case, that of a physician, a competent surgeon diagnosed an arterial embolism and was ready to apply a negative pressure apparatus; in the other, the initial venous thrombosis was massaged by an osteopath who managed to produce an arterial thrombosis in the popliteal artery. Such secondary arterial thromboses following a periphlebitis and lymphangitis around the thrombosed vein are rare, but may occur spontaneously. The arterial thrombosis is the result of a direct spread of inflammation within the common sheath.

The difficulty in differentiating arterial thrombosis from embolism is further increased by the fact that even surgical exposure, arteriotomy and a histologic section of the involved vessel may not clarify the picture. Thus an embolus may very quickly produce a secondary thrombosis both proximally and distally from the original plug. Furthermore, an embolus seriously interferes with the nutrition of the intima and creates a reaction of the vessel wall, which may be hard to distinguish from that of an arterial thrombosis. Even a postmortem examination may leave the problem unsolved, as a small auricular thrombus breaking loose and leaving a small rough surface on the lining of the auricle may be and has been overlooked.

In Table III my experience with acute arterial occlusions has been summarized.

TABLE III
DIAGNOSIS OF 39 CASES OF ACUTE ARTERIAL OCCLUSION

Embolism		*Thrombosis*	
Endocarditis	3	Buerger's disease	9
Coronary occlusion	2	Arteriosclerosis	8
Mitral stenosis	3	Diabetes	3
Postoperative	2	Postinfectious	3
		Posttraumatic	3
		Postphlebitic	1
		Unclassified	2
	10		29

In this group there are 2 cases erroneously diagnosed as embolism and proved to be

thromboses; one was a forty-two-year old woman, who was under treatment for multiple sclerosis, in rapid succession showing a brachial, a popliteal and a mild mesenteric occlusion. The exploration of the popliteal embolus revealed a widespread inflammatory lesion leading to multiple arterial thromboses and perhaps to the neurologic symptoms. The embolectomy was unsuccessful and a midthigh amputation had to be performed. She is alive after three years. The other case was postoperative, following a prostatic punch operation on a seventy-two-year old diabetic. He was seen four days after the vascular occlusion and died after an amputation. The obstruction was found to be due to a spreading infection from the pelvis to the iliac artery. Of the 27 cases that were finally classified as thromboses I know of at least 2 that proved to be of embolic nature; one was a forty-two-year old dentist who had a sudden vascular occlusion in one leg which was diagnosed as Buerger's disease because of the involvement of other vessels. Later he developed a cerebral and a coronary embolus, the postmortem showing a ball-valve thrombus in the heart. The other case, developed a bilateral vascular occlusion in the lower extremities. She has had previous premonitory symptoms, rheumatism and a pyelonephrosis. Following extraction of some infected teeth a sudden vascular occlusion occurred which was treated conservatively. Because of a definite heart murmur a diagnosis of bacterial endocarditis was made. She died refusing amputation. At autopsy, done in another hospital, no evidence of heart disease was found and diagnosis of Buerger's disease was made on the basis of the histologic picture at the site of occlusion, which, however, seemed doubtful. Re-examination of the records found a thrombosis of the renal artery on the side of infection from which a thrombus could well have broken loose if it extended to the aorta.

The differential diagnosis between arterial thrombosis and embolism has much more than academic interest. Recently Edgar Allen[7] has emphasized the difficulty of separating and reclassifying these two groups, especially on the basis of older histories, with incomplete data. Obviously in the cases of arterial thrombosis, the extraction of the clot would be quickly followed by a reformation and possibly an extension of the thrombus; and in the case of embolism the success of the operation rests mainly on the reactive changes in the intima and on the extent of secondary thrombosis.

With regard to the results of treatment, the collective statistics of Pearse reemphasize the original postulates established by Einar Key (Table IV). The time factor is

Table IV
Results of Embolectomy

	Per Cent Successful
First 10 hours	40
Second 10 hours	14
Third 10 hours	8
No successful cases after 48 hours	

Success is defined as a case in which circulation has been restored for at least one month. (Pearse. *Ann. Surg.*, 98: 17, 1933.)

of utmost importance in acute arterial occlusions, not only in cases suitable for embolectomy but also in those where adequate conservative treatment is followed. It might be well to consider briefly the early consequences of sudden arterial occlusion from which the utmost urgency of treatment can be readily derived.

When a larger artery is suddenly obstructed, the blood pressure must fall below the obstruction. This fall has been measured in the experimental animal by Melzner[8] who found that so long as the pressure remains above 20 mm. of mercury, no gangrene will develop; but below 15 mm. of mercury, a secondary thrombosis occurs below the ligature followed by gangrene.

Now this residual pressure is maintained by collateral circulation which in turn is dependent on cardiac output, on the site of obstruction and the state of collaterals. The only factor we can modify is the state of the collateral bed at the time of a

sudden occlusion, be it by ligature, embolism or thrombosis. The viability of the limb will depend upon the avalaible collaterals. It has long been supposed and frequently stated that sudden arterial occlusions produce a reflectoric vessel spasm. Allen and MacLean[9] have referred to the literature containing evidence. In the accompanying diagram (Fig. 2) I have attempted to show the pathways of such a reflex. The afferent arc is formed by the ordinary sensory nerves with which the vessels are well supplied. The impulse passes through the posterior root ganglion into the posterior horn from where it is relayed with the help of a short connector neuron to the lateral horn. Here are the ganglion cells of the efferent sympathetic fibers which are relayed in the sympathetic ganglionated trunk and then go by the way of the somatic nerves to the blood vessels. There is considerable experimental and clinical evidence for such a reflex. It can be interrupted by (1) novocaine block of the peripheral nerves, (2) spinal anesthesia, and (3) sympathetic ganglionectomy. None of these is especially suitable in cases of acute thrombosis or embolism, although the sympathetic efferents can be temporarily interrupted by a paravertebral injection of novocaine, the effect of which wears off in a few hours.

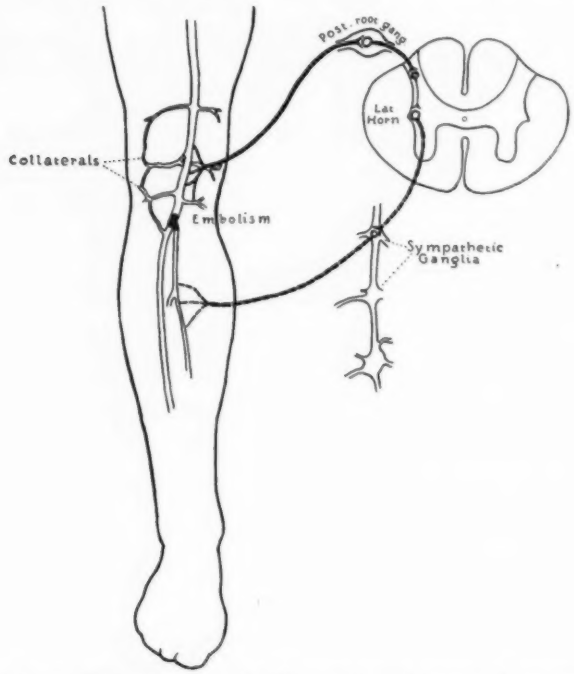

FIG. 2. *The reflex arc of collateral vessel spasm.* Sensory nerves are abundant in the coat of blood vessels. The afferent stimulus passes through the posterior root ganglion into the posterior horn; a short connector neuron relays the impulse to the lateral horn from where the efferent sympathetic fibers carry the vasoconstrictor impulse through the sympathetic ganglia to the periphery. A similar vasomotor reflex arc is postulated by Andrews (Ann. Surg., 85: 116, January 1927) except that he leads the afferent arc through the sympathetic ganglia and not through ordinary somatic nerves to the posterior root ganglion.

Because of the threatening gangrene that a widespread, concomitant vessel spasm may cause or accelerate, other simpler measures must be sought to overcome the collateral vasoconstriction. Of these, heat and rest are used most often. The temperature within the electric cradle, however, should be kept between 85° and 95°F.; more heat than that is painful and may invite gangrene. The patient's foot or arm should not be elevated as commonly taught, but on the contrary the affected extremity should be in the dependent position, thus raising the blood pressure in the dependent limb. The extremity should be wrapped in cotton which is held in place by a roller bandage. This may prevent pressure sores and helps to keep the extremity from cooling.

The most active measure at our disposal to relieve the reflectoric vessel spasm is the intravenous use of papaverine. Denk[10] suggested this and I have had enough experience with it to recommend its use most heartily. Papaverine produces a marked vasodilatation of the peripheral arteries. According to Macht it relaxes all types of smooth muscle without paralyzing them. In a group of 5 cases recently published,[11] marked and lasting benefit were obtained in 3; in 2 cases the drug was given too late to influence the appearance of gangrene. A dose of grain ½ may be given every four hours intravenously; to obtain maximal benefit papaverine should be given within the first six hours after acute arterial occlusion. The effect is then most

startling; the cold, cyanotic toe becomes pink and warm. In one instance, the skin temperature of the big toe was measured before and for thirty minutes after the injection and was found to rise approximately 2° in spite of a complete arterial occlusion of the common femoral artery. The drug does not produce an appreciable fall in blood pressure and pulse rate; it has a sedative and analgesic effect which makes morphine unnecessary. Its use in traumatic segmental vessel spasm and in arterial spasms accompanying phlebitis may be of considerable diagnostic and therapeutic value, but has not been reported thus far.

Another conservative measure to combat the concomitant vessel spasm is the alternating negative and positive pressure apparatus for patients with acute arterial occlusion as suggested by Herrmann and Reid.[12] The relief from pain is often quite dramatic and cases have been reported in which high arterial occlusions have been successfully treated without loss of any tissue. While I have had a number of such machines at my disposal since April, 1934 and have had the opportunity to treat 5 acute vascular occlusions with the suction apparatus, I have not been able to satisfy myself of a definite immediate benefit derived from its use.[13] It is true that at least 3 cases were submitted to this treatment after forty-eight hours and were hopeless. But one case has left a deep impression on me in regard to the relative merits of papaverine and negative pressure treatment. This fifty-one year old patient had a severe coronary occlusion with auricular flutter. Two weeks later the right foot became cold and cyanotic, but gradually improved. A few days later, or twenty-three days from onset, the right femoral artery became suddenly occluded. All toes showed discoloration and the skin began to blister. Passive vascular exercise with the Reid-Herrmann apparatus was started about twelve and one-half hours after the embolism, which seemed to relieve the pain but did not improve the circulation, although treatments were given from five to seven hours a day. When I first saw the patient twelve and one-half hours after the vascular occlusion, there were no pulses in the pedal and popliteal arteries, the oscillometric curves were absent as high as the midthigh and the histamine reaction was negative below the knee. A diagnosis of femoral embolism at the bifurcation of the deep and superficial femoral was made. Papaverine hydrochloride was started intravenously twice a day and continued for four days. Soon after the first injection a marked change took place in the color of the affected extremity; it became pink and the cyanosis of the toes disappeared. While the patient went through a mesenteric, a cerebral and another coronary occlusion, he is still alive eleven months after the embolism, with a sustained circulation of the affected limb.

In this instance the papaverine seemed to relieve the vessel spasm much more readily than the suction apparatus. The value of the latter, I believe, lies more in maintaining and gradually improving the gain in collateral circulation.

Recently Lehman[14] suggested the elevation of the environmental temperature by blankets and a heat cradle to overcome the vessel spasm of collateral channels following sudden occlusion of main vessels. This suggestion merits serious consideration and could be well combined with papaverine and passive vascular exercise.

In regard to the proper indications for a surgical removal of the embolus, it might be more convenient to define the conditions under which embolectomy is futile. Embolectomy is not indicated after forty-eight hours or even before that if there is a manifest gangrene nor in patients in whom the underlying disease is apt to be fatal shortly, as in septic endocarditis or terminal cardiac decompensation. It is seldom indicated on the upper extremity, but whether upper or lower extremity is involved, an hour can be safely used to see what heat and papaverine will do to relieve the vessel spasm. Obviously, the extent and duration of the occlusion and the rigidity

of the available collateral vessels sets a limit to the capacity of these conservative measures to aid collateral circulation. If this does not rapidly improve, embolectomy should not be delayed.

The site of the embolus can be determined fairly readily in most instances and the advisability of using opaque substances to visualize the obstruction is seriously doubted. In those patients often desperately sick, a simple exposure can be made under local anesthesia. Should the obstruction be at the aortic bifurcation or in the external iliac artery, an exposure of the femoral artery in Scarpa's triangle is the most advisable. The clot is then brought down by aspiration or, at the suggestion of Andrews,[15] with the help of a well fitting oiled catheter which acts as a piston. The technique of arterial incision and suture may be mastered in the animal but the entire equipment of special needles, finest Chinese silk, Carrel clamps must all be assembled ready for use to ensure a successful embolectomy.

Of the 10 cases that I have diagnosed as arterial embolism, 5 have been explored. The rest arrived either too late or responded to conservative measures. Of the 5, there were 2 each of popliteal and iliac occlusions, and 1 femoral. One popliteal occlusion, as mentioned previously, proved to be a thrombosis and required amputation. The other patient with popliteal occlusion lived a week after embolectomy with a restored circulation but died of a mesenteric embolism. The femoral and iliac occlusions were explored through an incision just below Poupart's ligament. The patient with the femoral embolus had a bacterial endocarditis and a week later threw another clot into the other leg. Further embolectomy or amputation was refused. The 2 cases of iliac embolism, both due to myocardial infarcts, survived. Thus 2 of 5 embolectomies were successful, a very modest result.

In the late cases arriving after forty-eight hours, an amputation should not be delayed if there is any chance of saving the patient's life. It takes considerable courage and optimism to handle these extremely handicapped patients, but the attitude of the family and the attending physician is often an additional problem. I have 2 patients living over two years, in each case a leg had been amputated because of an acute arterial occlusion and they are eloquent testimony of the value of not giving up the fight too soon. Naturally, a moribund patient should not be subjected to any further ordeal and the risk of amputating these limbs must be weighed against the possibility of saving the life of the patient.

Very little is known about the circulation of extremities that have survived the acute vascular occlusion without gangrene. I have 3 such patients in my material, one who developed an iliofemoral embolus following partial gastrectomy, another who had an arterial thrombosis of the femoral artery following pneumonia, and a third whose vascular occlusion followed a cholecystectomy. The limb of each of these patients is atrophic, pulseless, the muscles are contracted and fibrosed and the skin scaly and mottled. There is an intermittent claudication. Such patients are usually diagnosed as Buerger's disease, but a good history will elicit the origin of the sudden vascular occlusion. If the patients show a vessel spasm in these limbs, the origin of which is identical with that described as occurring in the acute arterial occlusions, a lumbar sympathectomy may be of great benefit. This has been done in one of these cases with the result that the collateral vessel spasm has been released, the limb lost its mottled cyanotic hue and walking has improved markedly. A detailed study of this group will be made later.

CONCLUSIONS

The study of the literature and my own material has led me to adopt the following attitude in cases of acute arterial occlusion:

1. An attempt should be made to differentiate between embolism and thrombosis, keeping in mind the possibility of traumatic

segmental vessel spasm and venous or lymphatic block with secondary vessel spasm.

2. One must try to localize the site of occlusion and determine on the basis of the patient's age, cardiovascular status and the known incidence of gangrene from occlusion at that site whether or not a gangrene is to be expected.

3. It is necessary to utilize vigorous conservative measures, including heat, dependent position, papaverine and negative pressure, if available, with the purpose of overcoming the concomitant vessel spasm.

4. Should these measures fail and equipment be available, an embolectomy is indicated, especially if the patient is seen within the first ten hours.

5. If these measures fail or if gangrene is present, amputation is indicated at the level of adequate circulation if there is a chance to save the patient's life.

6. A less passive attitude in regard to these peripheral vascular crises which are still being ignored or treated with indifference, will lead to an improvement in the mortality statistics of life and limb.

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ROLE OF FIBROUS TISSUE IN HERNIA REPAIR

SPECIAL REFERENCE TO INJECTION THERAPY*

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THE part played by fibrous tissue in the reparative process of wounds has been known since the dawn of pathology. Its importance, however, in the cure of certain pathological conditions has only recently received consideration. More than a century ago the injection of sclerosing solutions such as alcohol, iodine, cantharides, Lugol's solution and quercus alba were used in attempts to cure hernia, with the object of setting up an inflammatory reaction within the tissues, but the caustic effects were often so terrific that the treatment had to be abandoned. The injection treatment of hernia was first used by Velpeau in 1835, subsequently employed by Pancoast in 1833; Schwalbe and Heaton in 1877; Warren in 1881. Manely[1] published a monograph in 1893 and gave a general résumé of the clinical use of the injection method up to that time. Pina Mestra[2] perfected the modern method, and in 1927 reported more than 10,000 cases in which the treatment had been used with marked success. G. A. McDonald, Fairfield, Illinois, has used the treatment successfully for more than twenty-five years. In 1929 Mayer[3] reported a series of 2100 inguinal, femoral and umbilical hernias, treated with complete relief without recurrences after a period of twenty-five years, in 98 per cent of cases. In recent years many mild sclerosing solutions have been used, but only very recently has there been any concerted interest in the injection therapy of hernia.

To determine the process by which the cure is effected and to prove the utility of the procedure, Hall[4] in 1929 did some injections of hernia and reported experiments on monkeys at Bellevue Hospital, New York, with the injections of mild solutions into the abdominal wall which resulted in permanent induration.

Bratrud[5] recently reported on microscopic specimens made after injections of tannic acid solutions and thuga mixture all of which showed marked proliferation of fibroblasts dipping deeply between the muscle fibers. There was no infiltration of polymorphoneuclear cells or evidence of necrosis. More recently Rice[6] and his associates in the hernia clinic at the Minneapolis General Hospital, did some very interesting experiments on biopsy specimens obtained from patients, who for various reasons did not complete the treatment and were operated. Specimens were taken at intervals of five hours to forty-two days. Histologic sections revealed progressive stages at the end of fifteen hours; at the end of the fifth, eighth, fourteenth, eighteenth, and forty-second days. The tissues at the end of forty-two days was dense and resembled adult fibrous tissue. (Figures 1, 2, 3 and 4.)

A similar process takes place within the intima of an injected varicose vein, the lumen of the vessel being filled with a clot that firmly adheres to the vessel wall. The clot finally becomes organized into firm fibrous tissue, transforming the vein into a cordlike mass. In similar manner, injected hemorrhoids are followed by the development of fibrosis with obliteration of all the involved veins. As long ago as 1880, Billroth made the statement that if a solution could be obtained that created

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* Read before the Des Moines Academy of Medicine and Polk County Medical Society, May 26, 1936.

artificial proliferation of tissue, the radical cure of hernia would be solved.

Thus, the cure in all of these conditions is effected by deposits of fibrous tissue proliferation. In the case of hernia the deposits become firmly fixed within the rings and musculature of the canal, forming an efficient barrier against recurrence. The wearing of a truss during and following the treatment for some months tends to fortify the results. Bratrud believes that a truss should be worn for a few months following all operative procedures for hernia. Fibrous tissue formation following hernioplasty undoubtedly plays a much more important part in the cure than removal of the sac, replacement of the cord and the suturing process. The injection treatment of varicose veins and internal hemorrhoids is now acknowledged to be the method of choice in practically all cases. Hydrocele is no longer surgical, since injection of a mild sclerosing agent effects a cure in practically all cases. Injection treatment of hernias, however, has not received universal recognition by the profession; some of whom have pronounced it unscientific and dangerous. Clinical experiences prove that such allegations are unfounded.

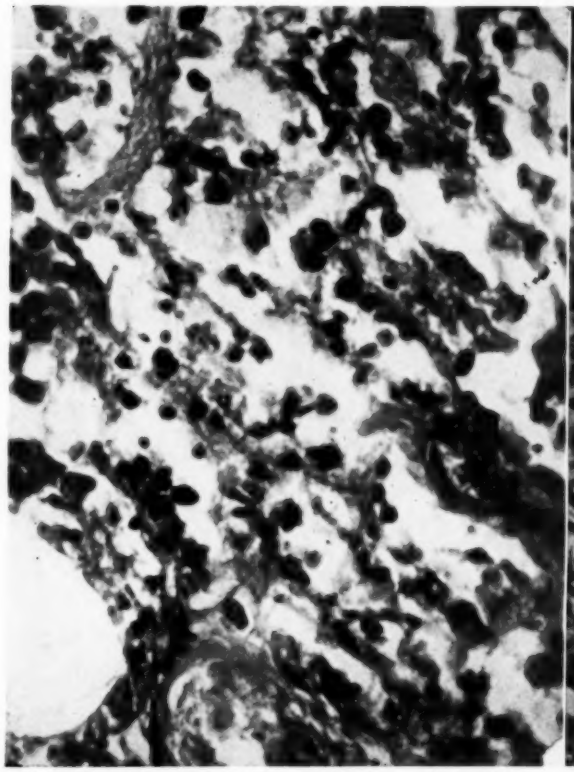

Fig. 1. Microphotograph from a section taken fifteen hours after the injection of the irritating solution. Polymorphonuclear cells, round cells and a few fixed connective cells. Magnification 800 ×. (*After Rice.*)

Fig. 2. Seven days after injection. Young fibroblasts and a few newly formed blood vessels predominate the picture. A few round cells but only an occasional polymorphonuclear cell can be seen. Magnification 200 ×. (*After Rice.*)

More than 1000 cases have been treated in the Minneapolis General Hospital and the University Hospital in Minneapolis, where the therapy has been in use for more than four years. No deaths have been reported following the treatment and but few untoward results have occurred. The treatment is being used by many representative surgeons and the technique is being demonstrated in surgical clinics in this country and abroad. In Volume II of Postgraduate Surgery,* now in preparation, St. George B. Delisle Gray F.R.C.S., Edenburg, contributes an article on injection of hernia. Ever since Halsted and Bassini perfected the operative treatment for the repair of hernia, its cure has been considered purely surgical, and until re-

* D. Appelton and Company.

cen tly has tended to divorce all other me thods of treatment.

It is admitted that the injection proce-

Fig. 3. Fourteen days after the injection. Young fibrous tissue with small slender nuclei can be seen. Fibers moderately abundant. An occasional round cell but no polymorphonuclear cells could be seen. Magnification 200 ×. (*After Rice.*)

Fig. 4. Forty-two days after injection. Fibrous tissue is abundant; fibers wavy. At the right of the section the proliferation of fibrous tissue does not appear to have progressed as far as at the left side of the picture. (*After Rice.*)

dure is not to be undertaken by anyone who is not familiar with every aspect of hernia. An accurate diagnosis as to the exact type of hernia is absolutely essential in each individual case and an intimate knowledge of the anatomy is necessary. One who has performed or assisted in numerous operations for hernia, and having accomplished the technique by actual observation can safely do this work. The needle sense acquired in practicing other injection therapy is a decided asset. Mayer has stated that the injection method requires fully as much skill as the radical operation, and an equal knowledge of anatomy. A review of the recent literature depicting the rationale of the method and results obtained, should readily convince anyone with an unbiased mind that the injection therapy of hernia is the method of choice in a large majority of cases. The treatment is ambulatory and may be done in the office, therefore, no hospital expense is incurred. No anesthetic is require as the treatment is practically painless, and the patient continues his accustomed avocation, except that of very heavy work. Any hernia that is fully reducible and can be retained by a truss may be successfully treated. Cases that are difficult to hold with a properly fitted truss, if put to bed for a few days, and injected daily, or every other day, may be treated successfully. By this time enough swelling results, which with the aid of the truss will hold the hernia safely within the abdomen. Two cases recently were treated in this manner, being injected every other day, and at the end of ten days the hernia was held securely. Ambulant treatment was then started.

It is not proposed that this treatment should supplant surgery, but the many thousands of people, who for one reason or another will not, or can not submit to operation and hospitalization, will gladly

accept this milder and less expensive method of cure.

Certain types of hernia are amenable only to surgery. Postoperative incisional hernias that have adhesions with incarcerated loop of bowel and no definite sac should be operated. Incarcerated or strangulated hernias, also hernias with undescended testicle, sliding hernias, and hernias that can not be completely reduced, should be operated. All indirect and direct inguinal hernias, femoral and umbilical hernias with a definite sac may be successfully treated. Slight incisional hernias that are reducible are also amenable to this therapy. One must have a clear conception of the object to be obtained in the eradication of hernia. The sac must be obliterated and closure of the opening through which the hernia first makes its appearance must be effected, by building a defensive network of adhesions to strengthen the fascial and muscular wall anterior to the internal ring. The primary object of this treatment is to set up an active seroplastic exudation, causing but little inflammation, with the formation of firm fibrous connective tissue proliferation, filling and obliterating the hernial tract.

Before beginning the treatment the patient must be fitted with a truss to be worn at least two weeks. Any good spring truss may be used but it must retain the hernia at all times. During the treatment the truss must be worn day and night, and for at least thirty days thereafter. It should then be worn during the day for ninety days or longer. Sponge baths may be taken during the first two weeks of treatment, after which the hernia may be held back with the fingers while getting in and out of the tub. At all times the truss should be removed and applied while lying down, the center of the pad resting over the opening through which the hernia first makes its appearance. It must be fitted by the physician who should perfect himself in its proper adjustments. In indirect hernia, trusses are almost invariably fitted too low, that is, medial to the internal ring. Coöperation at all times is necessary and this must be impressed upon the patient at the beginning, otherwise failure is almost certain. As 90 per cent of the trusses being worn I have found to be inefficient, a department of truss fitting should be added to the curriculum of all medical schools.

INGUINAL HERNIA TECHNIQUE

Various techniques are described, but all agree that uniformly successful results are obtained only when the solution is injected beneath the aponeurosis into the region of the inguinal rings and into the canal. Of course the internal ring must be accurately located. It may be found approximately, by drawing a line from the anterior superior spine of the ilium to the tubercle of the symphysis pubis; the ring being located halfway between these two points and a finger breadth above it. The patient is placed upon the table which is tilted to a slight Trendelenburg position and the hernia reduced. The pubic hair may be clipped, but not shaved. The area is thoroughly mopped with 70 per cent alcohol and the site for puncture is marked with a dash of merthiolate or iodine solution. The needle is then inserted at a point about 2 cm. above the ring at an angle of about 40° downward and caudilward, until the point reaches the area of the internal ring, which will be found from 1 to 3 inches from the point of insertion, depending upon the thickness of the abdominal wall. Before making the injection, the plunger of the syringe should be slightly withdrawn to make sure the point of the needle is not resting in a vein or in the peritoneal cavity. If no blood is aspirated and negative pressure is shown in the syringe, the injection may proceed. If acute pain attends introduction of the needle, it should be withdrawn slightly and its direction changed, as the point may be in contact with a nerve or resting in the cord. Only a fraction of a drop should be injected at first. If no pain develops the injection may continue very slowly, drop by drop. When the injection is completed the needle is removed and pressure for a few moments is applied over the site of the needle puncture. After ten or fifteen minutes a thin layer of sterile gauze is placed over the puncture

and the truss is applied while the patient remains in position; he is then raised to his feet. Injections are repeated at intervals of three to five days if no marked soreness develops. Cases ordinarily require from six to twelve injections, the last few being distributed along the hernial canal and within the external ring. More injections usually are required in direct than in indirect hernia. Femoral hernia requires fewer treatments, three to five usually being sufficient. Any after-pain may be controlled by packs and hot water bottle.

SOLUTIONS USED

I shall describe only the solutions readily available and of known merit. The ideal solution, one that will require but one or two series of injections to complete the cure, is yet to be found. Mayer's solution consists of zinc sulphate 1 dram in phenol crystals 6 drams, glycerine 4 fluid ounces, aqua cinnamomi 1 fluid ounce, fluid extract pinus canadenisis (dark) 5 fluid drams in chemical pure redistilled water sterilized 2 fluid ounces, and 8 to 16 minims are injected every four to seven days. Proliferol is a mixture composed of tannic acid, thymol and benzyl alcohol in 95 per cent alcohol. Two c.c. of novocaine is injected, and from 2 to 5 c.c. of the solution is deposited through the same needle. McDonald's solution, No. 2, is composed of Lloyd's specific tincture of thuga 25 parts, alcohol 25 parts, phenol crystals 50 parts. It should set twenty-four hours and then be filtered. The usual dose is 5 minims at each injection. Sylasol, a compound of sodium salts of fatty acids of psyllium seed has recently been introduced. While comparatively little clinical use has been made, experiments have shown that it produces fibrosis and it requires no anesthetic.

COMPLICATIONS

Inflammation and hydrocele of the cord has been noted in a few cases. Hot packs brings relief when pain is present, and the swelling subsides within a few weeks. In a few instances abscess has been reported following the treatment, probably due to error in technique or in the preparation of the field. The fear of other complications has been mentioned, such as sterility, strangulation, epididymitis, atrophy of the testicle and peritonitis. None of these complications, however, have been reported. It is too early to realize the effect if any, the treatment may have on sterility. Atrophy of the testicle is occasionally noted in the examination and if present, it is important to mention the condition in the record before treatment is begun. Bratrud[7] states that in over 700 cases treated, he has not observed atrophy of the testicle following treatment.

RECURRENCES

Recurrences following the injection treatment appear to be less frequent than following operation. Some of the earlier enthusiasts evidently have been over optimistic in placing recurrences too low. Deaver, at the Interstate Congress in Minneapolis in 1930, reported 8 per cent known recurrences of hernia in his clinic. Statistics according to Bratrud[7] show 17 per cent surgical recurrences and he admits 7 per cent returns in his clinic after injections, but any slight impulse requiring reinjection was considered a recurrence. Most of these cases should be permanently cured by reinjection. It is well known that varicose veins that have partially recanalized after the injection treatment are permanently obliterated after reinjection.

CONCLUSIONS

1. The injection treatment is safe in the hands of a competent operator, and in selected cases it is at least equally successful as hernioplasty. There is danger of complications, however, if details are not strictly followed.

2. The coöperation of the patient is essential in the wearing of a truss. It must be fitted accurately by the physician and hold the hernia in at all times.

3. The method saves economic loss in that the patients' earning power is not curtailed and the total cost is less.

[7] Bratrud, A. F. *Jour. Iowa State Med. Soc.*, 25: 591–597 (Nov.) 1935.

MANAGEMENT OF RUPTURED APPENDIX*

AN ANALYSIS OF 50 CONSECUTIVE CASES

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OBSERVATION in the literature of a universally high mortality rate with an appalling incidence of morbidity in late cases of appendicitis stimulated the writers to examine carefully their own records of these cases for the past few years. The high death rate observed indicates unquestionably the seriousness of this problem. Every surgeon is aware that no type of case requires more mature thought and judgment than does the ruptured appendix.

No attempt will be made from the analysis of this small series to offer either a new or standard method for the management of all late cases of appendicitis. Only by establishing proved methods of treatment and discarding condemned ones can the care of these cases become standardized and the results improved. However, there are definite reasons why observations based upon a small personal series of private cases should be relatively important. Often in large series of cases the observations are based upon the results obtained by a number of surgeons, or operators, of varying ability and judgment. The aftercare of these cases in large clinics or hospitals is far too frequently left to an inexperienced house staff when this care is just as important, and in some instances, far more so than the actual operation. In this, as in similar series, the same operating team was present in each case, the final judgment and decision rested with the senior surgeon and the postoperative treatment was carefully and constantly supervised by him.

Since January 1, 1933 there have been admitted to this surgical service 156 cases diagnosed as appendicitis. Of this number 50, or 32 per cent, were found to have macroscopic pus either free or localized in the peritoneal cavity; 5 of these showed actual gross perforation of the wall of the appendix and 11 appendices were not removed. In the remainder, peritoneal involvement presumably occurred by extra-serosal extension.

This paper deals primarily with the management of late appendicitis. However, several interesting points not related to treatment were demonstrated in this analysis. Thirty-five cases of the 50 took or were given one or more purgatives during their illness. Forty-three were from rural districts. In 20 cases a physician was called at the onset of the pain. The duration of subjective illness in the entire series ranged from one to fourteen days with an average of 4.5 days. In only 7 cases could any definite history of previous attacks of appendicitis be elicited. Thirty-two cases occurred in males; 18 in females. The ages ranged from four to fifty-eight years with an average of twenty-eight years. Total white blood cell counts ranged from 2500 to 39,750, with an average of 17,500. The neutrophile count averaged 84 per cent in this series. Hospitalization extended from ten to fifty days with an average of nineteen days.

The significance of 70 per cent receiving purgatives is well understood and should need no further discussion. We noted with regret that 6 were administered purgatives by the attending physician. The fact that 40 per cent of these cases were seen by physicians at the onset of illness by no means indicates that they were always to blame for the delayed operation, for many of these physicians have proved themselves

* From the Surgical Service of E. Dunbar Newell, M.D., F.A.C.S. Read before the Chattanooga and Hamilton County Medical Society, February, 1936.

repeatedly to be acute diagnosticians. The majority of rural patients refuse hospitalization and operation until they are sick enough to realize that the physician's judgment is better than their own. In a number of other instances bizarre symptoms obscured the diagnosis; in 4 the initial symptom was severe diarrhea; in one the symptoms and signs simulated those of coronary occlusion and another case complicated by severe auricular fibrillation was diagnosed early as digitalis poisoning.

Every one of the 50 late cases was operated. There were 2 deaths; both occurred in children under nine years of age with ruptured appendices, rapidly spreading peritonitis and fulminating toxemias. This gives a mortality in the entire series of 1.2 per cent and in the 50 late cases of 4 per cent. These figures compare favorably with those observed in the literature. We are fully cognizant of the successful and much discussed delayed surgical treatment of neglected appendicitis. Each patient in this series was carefully and critically individualized. We have endeavored at all times to base our treatment on proved physiologic principles. It seemed to us that immediate surgical intervention offered every one of these patients their best chance for recovery. It is possible that the 2 cases which succumbed following surgery were the ones in which delayed treatment would have been successful. That question will remain unanswered for us until we observe the results of conservatism in similar types of pathology. We sincerely hope conservatism in selected cases will materially improve mortality statistics in this dread condition.

The type and technique of operation is the most important item in the surgical management of the ruptured appendix. Improper operating room procedures forecast heroic postoperative measures, multiple disastrous sequela and disheartening, fatal results. In the 50 late cases 46 received straight ether or combined ether, nitrous oxide and oxygen anesthesia; 2 cases received spinal anesthesia because of severe coryza and one patient indicated a preference for this type of procedure and local infiltration anesthesia was used in the case complicated by severe auricular fibrillation. Spinal anesthesia offers several advantages over general anesthesia where intestinal relaxation is essential, however, we are convinced that this is offset by definite limitations and indications in private practice. As will be shown later, postanesthesia difficulties have not made it necessary for us to find a substitute for properly administered inhalation anesthesia.

The incision is never made until the patient is thoroughly relaxed. It is felt that the trauma attendant to operating upon a tense straining abdomen with threatened evisceration does the patient far more harm than a few extra minutes spent under an anesthetic.

The majority of these cases were opened by a 2½ to 4 inch vertical incision in the right semilunar line as this type of incision affords excellent exposure directly over the operative field and prevents needless moving and packing off loops of normal bowel to reach the diseased area. These advantages preclude the use of harmful rigid, metal retractors in the wound. Our results show 4 postoperative hernias in the 50 badly infected cases; certainly too small a percentage to blame the type of incision used. We feel that an operation with the minimum of intestinal trauma followed by a hernia in a live patient is much better than a fancy incision in a dead patient. When it is thought that a definite localized abscess has formed around the involved appendix, a small vertical incision is made as near the anterior superior iliac spine as feasible, so that these abscesses may be drained extraperitoneally. No hesitation is felt in lengthening the incisions in either, or both, directions to afford adequate exposure.

The question of whether to remove the appendix or simply drain depends entirely upon the individual type of pathology present. In 11 cases the appendix was not even visualized; of which 7 were classified

as true localized abscesses and treated by adequate extraperitoneal drainage, the other 4 evidencing generalized peritoneal involvement. In these the abdomen was filled with thin, watery pus and the patients were critically ill before subjected to surgery. The 2 patients who succumbed are included in this last group. It was felt that manipulation necessary to remove the appendix was unwarranted, therefore, only peritoneal drainage was instituted. Realizing that drainage of the entire abdominal cavity was impossible we attempted to drain those areas most likely to afford serious trouble later, viz: (1) the pelvis, (2) along the ascending colon to the right kidney fossa, and (3) the appendiceal area. In 9 other cases peritoneal drainage was instituted for definite reasons. They presented a total necrosis of the appendix with more or less involvement of the cecal wall, edema of the surrounding peritoneum and a white plastic exudate involving the appendiceal bed, omentum, adjacent intestines, and, at times, the bladder and Fallopian tube. It would be optimistic to suppose even a very healthy peritoneum could absorb this necrotic exudate. The drainage material used was soft rubber without wicks. The position of the drains except in the necrotic appendiceal area depended upon the region showing the most involvement. Adequate drainage in these as well as in the abscess cases is very important. The drains were removed by withdrawing a small portion with each dressing, after three to fifteen days, the average being nine days, the final removal was indicated by the amount and character of the drainage and the febrile reactions of the patient. It is much better to allow these drains to remain a few days too long than to face the problem of having to replace them; this occurred once in this series.

In the remaining 30 cases the peritoneal cavity was closed without drainage.

In the cases in which the appendix was removed, it was done with as little attendant trauma as possible. The stump was tied and inverted in the majority of instances. When excessive necrosis of the appendix or adjacent cecal wall prevented invagination, merely tying the stump securely and cauterizing with iodine sufficed.

No attempt was made in any case to cleanse the peritoneum by the use of a suction apparatus. Neither was the pus or intestinal contents flushed from the abdominal cavity by various fluids. In every instance it was felt that the non-traumatized peritoneum and proper supportive postoperative measures could adequately care for the infection present.

The fascia and skin were closed in all cases in which the peritoneum was not drained. As a rule small rubber tissue drains were placed under the skin sutures. In the drainage cases the skin was not closed. A few loose sutures were placed in the peritoneum and fascia to prevent evisceration and to promote healing and the wounds dressed with sterile vaseline gauze. We attempted without success in numerous cases to sterilize these wounds and properly shield them from all infected material. Only 9 cases healed by primary intention.

At no time have we been forced to perform an enterostomy following these operations and have had no fecal fistula develop. Severe ileus is almost always a result of operative trauma or unwise postoperative care. An attempt was made here to avoid these mistakes. Therefore, no reason was apparent for performing a cecostomy by inserting a tube in the appendicial stump. This procedure however is very successful in the hands of others and merits consideration.

Proper postoperative care is second only in importance to the operative procedure and at times is even more important. All of these cases have a potential adynamic ileus but it can be avoided by proper prophylaxis. Its persistence depends upon the operative trauma, the amount and character of infection present and the too early administration of liquids by mouth. Extreme gentleness during operation, prevention of spread of peritoneal infection by

contamination and withholding fluids or food in the intestinal tract are all essential factors. Morphine was given liberally to adults in ¼ or ⅙ grain doses, hypodermatically, every four to six hours and children under twelve years of age received codeine. The morphine was administered as an analgesic, and because it has been shown to increase definitely intestinal tone and prevent distention. Dry heat in the form of an electric pad or heat tent was applied to the abdomen. It has been demonstrated that the resulting dilatation of somatic vessels causes contraction of the splanchnic vessels, thus decreasing the tendency for intestinal stasis. These patients were not allowed anything by mouth for at least seventy-two hours following operation. If at this time they showed no nausea or distention, small amounts of hot water were given, followed later by hot tea and broth with all fat carefully removed. It was noticed repeatedly that the early administration of cold or sweetened drinks invariably produced nausea, vomiting and distention. Should these patients show the least tendency to distention or nausea a duodenal catheter was at once inserted intranasally and active suction instituted. These tubes cause little inconvenience and may be left in situ for a number of days if necessary. No patient was allowed to go more than eight hours without voiding, and catheterization was done more often if necessary. Cystitis was not one of the complications occurring in this series. Proctoclyses were not given as their value is questionable as far as fluid replacement and nutrition is concerned and they certainly disturb the gastrointestinal tract. Enemas were not administered until the patient definitely overcame the ileus present and the intestines showed a tendency to onward peristalsis. This varied from five to eleven days.

Dehydration and lack of food, two most important items, were combatted by the liberal use of subcutaneous and intravenous administration of physiologic salt solution containing dextrose. We prefer the intravenous method because it is less painful and more efficient. Both Hartmann's solution and plain sodium chloride were used with the destrose concentration varying from 5 to 15 per cent, according to the requirements of the individual patient. One patient in this series received absolutely no other food or fluid for twelve days except the intravenous and subcutaneous injections. Enough fluids were administered every twenty-four hours to cause the patients to excrete at least 1000 c.c. of uring during a corresponding period of time.

The care of the wound is most important to the welfare of these patients. Regardless of what protective measures or antiseptics are used, the wound is always potentially infected. Forty-one of the incisional wounds in this series drained profusely; 9 healed by primary intention; 2 cases required the use use of Dakin's solution for a few days because of excessive necrosis. In one instance the entire contents of the right inguinal canal and the right testicle sloughed off following a cellulitis of the abdominal wall. Abdominal wall infection can become a most formidable complication unless carefully managed. The wounds were dressed daily, using sterile pertrolatum and a mild antiseptic. Secondary abdominal wall abscesses were opened usually through the original wound. Cellulitis was treated by hot moist compresses. All infections responded readily to proper drainage. Healing time was from ten to ninety days, with an average of twenty-nine days. The wound edges were drawn together with sterile adhesive after the cessation of drainage and a secondary closure was necessary in only one case.

To us the most gratifying portion of our analysis was the relatively rare occurrence of serious complications and sequela and we strongly urge the prophylactic treatment of these conditions. The gentle operation, careful and physiological postoperative measures, constant attention to the wound and a well regulated regime for the general well-being of the patient.

In only one instance, in a patient with severe diarrhea, was it necessary to open a residual abscess. The abscess formed just above the bladder and was easily opened under local anesthesia. Two cases developed some tenderness on pressure over the twelfth right rib and one case complained of pain and tenderness along the descending colon. These evident inflammatory processes subsided with expectant treatment. In 2 cases it was necessary to incise secondary abscesses of the abdominal wall. One case developed a mild unilateral parotitis on the fourth postoperative day which subsided spontaneously with the application of heat. Prevention of excessive dryness of the mouth and attention to oral hygiene will usually obviate this condition. These patients were all instructed the first twenty-four hours postoperatively to take ten deep breaths every hour. Their position was changed frequently. One case developed a thrombophlebitis of the right saphenous vein which was treated only by absolute immobilization. There were 2 cases evidencing febrile reactions and a cough with frank bloody sputum lasting several days, indicating probably pulmonary emboli. Hospitalization was not extended in these cases and no permanent lung damage could be demonstrated. One patient developed a frank pulmonary abscess which ruptured into the pleura and into a large bronchiole. This abscess was drained externally by a hard rubber tube placed between the ribs and into the cavity. The lung expanded satisfactorily following spontaneous closure of the bronchial fistula. One patient developed a severe bronchorrhea and atelectasis of the upper right lobe of the lungs, probably caused by a mucous plug in a bronchus but no active treatment was required to overcome the condition. This one complication we feel is directly due to inhalation anesthesia. One case had a moderate secondary hemorrhage from the bottom of the wound on the fourteenth postoperative day, requiring careful packing for several days. The case with the pulmonary abscess developed acute dilatation of the stomach on the thirtieth postoperative day. He had been on solid diet for fifteen days and we feel the condition was probably precipitated by severe pain from the drainage tube between his ribs. He was immediately relieved by gastric lavage and drainage. The case operated on when five months pregnant aborted on the fourth postoperative day as was expected.

CONCLUSION

The writers have attempted from this analysis of a small series of cases of late appendicitis to determine their own mistakes and misfortunes. An effort has been made to justify the use of certain physiological methods of management found successful and to condemn other methods not successful. Hope is expressed that detailed analysis of many such cases will offer a solution to this most important surgical problem, the management of the ruptured appendix.

NEOPLASMS PRIMARY IN DIVERTICULA OF URINARY BLADDER

REPORT OF 5 CASES

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PRIMARY carcinoma of the urinary bladder frequently extends into and involves a diverticulum of the bladder but a primary neoplasm in a diverticulum is a rather infrequent finding. The following 5 cases of neoplasms which were primary in and confined to a diverticulum of the bladder have occurred in a series of 221 cases which have been seen at the Cleveland Clinic.

CASE REPORTS

CASE I. A man, seventy-four years of age, entered the Clinic on May 25, 1925, complaining of blood in the urine. He had been perfectly well until five weeks before our examination with the exception of nocturia two or three times, dribbling, diminution in the size of the stream, frequency and bright red blood in the urine. The general examination was negative with the exception of a grade 1 enlargement of the prostate which was movable and firm in consistency.

Laboratory Findings. Examination of the blood showed 3,620,000 red cells, 6900 white cells and 65 per cent hemoglobin. The blood sugar was 110 mg. and the blood urea was 21 mg. per 100 c.c. The Wassermann reactive was negative. The urine was alkaline in reaction and contained a trace of albumin but no sugar. The specific gravity was 1.017. Numerous red blood cells were present and there were some white cells. A roentgenogram of the kidneys, ureters and bladder revealed no abnormalities.

Cystoscopic Examination. The bladder capacity was 300 c.c. The cystoscope was introduced with ease and neither ulcers nor stones were seen but a diverticulum containing a tumor mass was found on the left side of the bladder. This presented a very unusual picture of a growth in a diverticulum. A cystogram showed a large irregularly outlined diverticulum on the left side of the bladder.

Clinical Diagnoses. Primary tumor in a diverticulum of the bladder; hypertrophy of the prostate.

Operation. Under general anesthesia the diverticulum was completely removed extravesically and the bladder closed without drainage. A rubber tissue drain was inserted retroperitoneally at the site of excision of the diverticulum. The ureter did not require reimplantation.

The patient's convalescence was uneventful.

Pathological Diagnosis. Papilloma in a diverticulum of the bladder.

One month after discharge from the hospital, the patient died suddenly, the cause not being determined.

CASE II. A machinist, sixty years of age, entered the Clinic April 2, 1930, complaining of bladder trouble. One month previously blood had been noted in the urine. Although the bleeding had been somewhat profuse at first, it subsided for a few days. Four days later, the urine again became quite bloody and bleeding was so profuse that a transfusion was required, following which the patient was admitted to the Clinic.

No familial history of blood diseases or cancer could be elicited.

Physical examination revealed an apparently ill man. The pulse rate was 130 and the blood pressure was 120 systolic, 80 diastolic. The general examination was essentially negative with the exception of a palpable globular mass above the symphysis which was due to the distended bladder. There was a grade II enlargement of the prostate which was firm and not fixed or tender.

Laboratory Findings. Examination of the blood showed 3,100,000 red cells, 12,100 white cells and 50 per cent hemoglobin. The blood urea was 39 mg., creatinine 1.0 mg. and sugar 98 mg. per 100 c.c. The Wassermann test gave normal findings. The urine was found to be acid in reaction with a specific gravity of 1.010.

Albumin was present but there was no sugar. There were many red blood cells and a few white cells.

Due to the profuse bleeding, a cystoscopic examination was not made and following a blood transfusion of 750 c.c. of whole blood, operation was performed.

Operation. Under nitrous oxide anesthesia, the bladder was opened and numerous clots were evacuated. Just above and lateral to the right ureteral opening was a diverticulum 6 cm. in diameter with quite a large orifice containing a small pedunculated tumor attached to the base. This had the gross appearance of a benign papilloma. A biopsy was done and the papilloma was thoroughly fulgurated. The incision in the bladder was completely closed without a drainage tube and the skin closed with clips.

Postoperative convalescence was uneventful and the patient was discharged from the hospital seventeen days after operation.

Pathological Diagnosis. Benign papilloma of a diverticulum of the bladder.

Three months later the patient was symptom free. However, cystoscopic examination revealed a small papillomatous growth just inside the diverticulum on the lateral wall. This was thoroughly fulgurized. An intravenous urogram showed no abnormalities.

The patient was seen two weeks later and sloughing was present in the area previously fulgurized. At the outer edge of this area was a small papilloma which was also fulgurized.

Three months later, cystoscopic examination revealed no evidence of recurrence and the patient was free from symptoms.

Death occurred two years later from cerebral embolism.

Case III. A salesman, sixty-nine years of age, was admitted to the Clinic, April 11, 1932, complaining of blood in the urine and nocturia. For five or six years progressive difficulty in voiding had been noted; nocturia had been present four or five times and frequency occurred during the day. A history of intermittent attacks of hematuria during the preceding year was elicited. At times, the urine was bright red with intervals when it was quite clear. For two years considerable burning during micturition had been experienced. Dribbling and more frequent attacks of hematuria had occurred during the preceding few months.

No familial history of cancer or blood diseases was elicited.

Physical examination revealed a well developed and nourished man who weighed 170 pounds. The blood pressure was 170 systolic, 90 diastolic, and the pulse rate was 90. The left testicle was atrophic and there was a grade II enlargement of the prostate which was firm and not fixed or tender.

Laboratory Findings. Examination of the blood revealed 3,862,000 red cells, 9000 white cells and 80 per cent hemoglobin. The Wassermann reaction was negative. The urine was acid in reaction, loaded with red blood cells and contained many white cells. Some albumin but no sugar was present. The blood urea was 45 mg. and the blood sugar 101 mg. per 100 c.c. The phenolsulphonphthalein test showed 48 per cent excretion at the end of the second hour.

Cystoscopic Examination. The cystoscope was introduced with some difficulty, and 300 c.c. of bloody urine was removed from the bladder. Repeated washing of the bladder left a clear field. At the point at which the left ureteral orifice should be seen, there was a distinct hog-back type of elevation. When the bladder was distended further, a large cavernous opening came into view which apparently led to a large diverticulum. All the bleeding seemed to come from this diverticulum, and there were some shaggy strands of clots hanging from the orifice.

Diagnoses. Hypertrophy of the prostate; diverticulum of the bladder with a question of the presence of a tumor.

The cystoscopic examination was repeated the following day. A catheter was inserted into the diverticulum and then filled with 30 c.c. of skiodan. A roentgenogram showed a filling defect in the diverticulum and a cystogram likewise showed the same defect. (Fig. 1.)

Final Diagnoses. Hypertrophy of the prostate; primary tumor in a diverticulum of the bladder.

Operation. Operation under spinal anesthesia was performed April 27, 1932. Careful inspection of the interior of the bladder revealed a large diverticulum filled with blood clots in the region of the left ureteral orifice which measured 0.5 cm. in diameter. A narrow gauze tape was gently packed into the diverticulum which was removed extravesically. Since the ureter could not be reimplanted into the bladder, it was ligated. The opening into the

bladder was closed in the usual manner and the incision in the bladder united.

The patient died from a pulmonary embolism six days after operation. Autopsy was refused.

Pathological Diagnosis. Carcinoma, primary in a diverticulum of the bladder.

Case IV. A man, fifty-nine years of age, entered the Clinic on June 12, 1934, complaining of frequency of urination and hematuria. For the past three years he had noticed moderate frequency and nocturia. In the preceding year the nocturia had increased from once to four times. Six months before entering the hospital the patient first noticed blood in the urine, and this was present for three days. Numerous clots were passed for a few days after the bright red blood disappeared. Since that time, painless bleeding had occurred on several occasions and usually lasted from two to three days. Just previous to our examination, however, the bleeding had been more profuse and dribbling and a progressive diminution in the size of the stream had been noticed. No familial history of cancer, tuberculosis or blood diseases could be elicited.

Physical examination revealed a rather obese man who weighed 185 pounds. The blood pressure was 160 systolic, 96 diastolic, and the pulse rate was 72. The general examination was essentially negative with the exception of grade II enlargement of the prostate which was soft, movable and not tender. A roentgenogram of the kidneys, ureters and bladder revealed no abnormalities.

Laboratory Findings. Examination of the blood revealed 5,100,000 red cells, 10,350 white cells and 91 per cent hemoglobin. The Wassermann reaction was negative. The urine was alkaline in reaction and contained many red blood cells as well as white cells. Albumin was present but there was no sugar. The blood sugar was 133 mg. and urea 33 mg. per 100 c.c. of blood. The phenolsulphonphthalein test showed 58 per cent excretion in two hours.

Cystoscopic Examination. The cystoscope was introduced with ease. Considerable cloudy urine was present in the bladder which was diffusely inflammed. Just lateral to the left ureteral orifice, a small diverticulum was seen and upon inserting a catheter into it, bleeding became quite profuse. Twenty c.c. of a 16 per cent solution of sodium iodide was injected into the diverticulum and a roentgenogram visualized the diverticulum excellently.

Operation. A small diverticulum just above and outside the left ureteral orifice was found which contained a small papillary growth. The diverticulum was easily inverted into the bladder and completely excised.

The postoperative convalescence was uneventful.

Pathological Diagnosis. Carcinoma of a diverticulum of the bladder.

The patient is free from symptoms and cystoscopic examination shows no recurrence one and one-half years later.

Case V. A man, fifty-five years of age, entered the Clinic December 9, 1935, complaining of hematuria. In June, 1934, he first noticed blood in the urine. A physician was consulted and diagnoses of diverticulum of the bladder and hypertrophy of the prostate were made. Bladder irrigations were given and the bleeding stopped for two days. The patient was then free from bleeding until July, 1935. Following that time, bleeding had occurred more frequently and ten weeks previous to our examination, irrigations were again given. Nocturia had increased gradually as had frequency and dribbling.

Physical examination revealed a well developed man who weighed 163 pounds. The blood pressure was 110 systolic, 76 diastolic and the pulse rate was 72.

The general examination was negative except for a grade II enlargement of the prostate which was firm and not fixed or tender. Above the prostate was a small, hard, fixed, immovable mass, 6–7 cm. in diameter, which simulated an extensive carcinoma of the floor of the bladder.

Laboratory Findings. Examination of the blood showed 4,900,000 red cells, 6900 white cells and 90 per cent hemoglobin. The urine was alkaline in reaction, contained many red blood cells, some white blood cells, a moderate amount of albumin but no sugar. The blood urea was 30 mg. and the blood sugar was 84 mg. per 100 c.c. The phenolsulphonphthalein test showed 55 per cent excretion at the end of the second hour.

A roentgenogram of the kidneys, ureters and bladder showed no abnormalities.

Cystoscopic Examination. The cystoscope was passed with ease. The bladder capacity was diminished and distention of the bladder with 125 c.c. of solution produced discomfort. The bladder wall was moderately inflamed.

The left ureteral orifice was not visualized but at this site was an opening into a large diverticulum. A catheter was passed and a roentgenogram showed it to be coiled up in a large diverticulum.

An intravenous urogram was made which revealed a diverticulum of the bladder containing a large, irregular mass, thought to be a carcinoma.

On the following day, 5 c.c. of indigocarmine was injected intravenously and the solution was seen to come from the right ureteral orifice in four and one-half minutes and in five minutes from the opening of the diverticulum on the left side of the bladder. The ureteral catheter was again passed into the diverticulum and 30 c.c. of skiodan injected. A roentgenogram showed the same filling defect in the diverticulum that was seen previously. (Fig. 2.) At this examination, blood could be seen coming from the orifice of the diverticulum.

An intravenous urogram showed both kidneys functioning well. The left ureter emptied into a large diverticulum of the bladder which showed a constant filling defect. (Fig. 3.)

Diagnoses. Hypertrophy of prostate; primary tumor of a diverticulum of the bladder.

Operation. Under spinal anesthesia a large diverticulum was removed extravesically and the bladder closed in the usual manner. (Fig. 4.)

The patient had a pulmonary embolism on the sixth postoperative day but recovered and was discharged from the hospital January 20, 1936.

Pathologic Diagnosis. Papillary carcinoma of a diverticulum of the bladder.

REVIEW OF LITERATURE

Targett[1] apparently was the first to report a primary neoplasm in a diverticulum of the bladder. In 1896 he examined 7 specimens in which malignant disease of the bladder and diverticula were associated and in one instance found that the neoplasm, an epithelioma, was confined to the diverticulum. In his review he included one case reported earlier by Williams.[2] A diagnosis of sarcoma had been made but Targett terms this as incorrect; however, Le Comte,[3] in his review of all neoplasms primary in diverticula includes this case reported by Williams. In 1896 Fenwick[4] reported a case and in 1909, Perthes[5] and Young[6] each reported examples of this condition. Buerger[7] in 1913 added another case and Thomas[8] in 1916 cited 3 instances of neoplasm confined to diverticula of the bladder. In 1924, Negro and Blanc[9] reported a papilloma primary in a diverticulum and Harris[10] reported an advanced carcinoma in a diverticulum. In the same year, Judd and Scholl[11] reviewed 133 cases of diverticula of the bladder which had been seen at the Mayo Clinic and found that carcinoma of the bladder and diverticula were present in 10 cases. In 4 of these, the carcinoma was primary in the diverticulum. Five years later, Hunt[12] reviewed this same series of 10 cases and found that evidence of secondary involvement of the bladder immediately adjacent to the orifice of the diverticulum was present in all except one in which the tumor was confined to the diverticula. He reported 4 of his own cases of primary epithelioma confined to a diverticulum, and added a case in which Judd had performed the operation in 1916. Deming[13] in 1927 reported a primary carcinoma in a diverticulum and in 1928, Lowsley and Gutierrez[14] reviewed 54 cases of diverticula of the bladder and found 3 instances in which tumors were primary in diverticula. Of these, 2 were benign papilloma and the third was a carcinoma. In 1929, Kretschmer and Barber[15] collected 20 cases of carcinoma in diverticula of the bladder and added another of papillary carcinoma. In the same year, Teel[16] cited 3 instances in which a tumor appeared to originate in the lining of a diverticulum. In 1930, Ewell[17] and Rathbun[18] each presented a case, the latter being a diverticulum which contained a carcinoma of cloacal origin. In 1931, Peacock and Corbett[19] collected 11 additional cases bringing the total number to 31. Stewart and Muellerschoen[20] cited the reports of Gill[21] and Hicks[22] and added a case of carcinoma of a diverticulum in which autopsy revealed extensive metastasis. In 1932, Le Comte[3] reported a collected series of 41 neoplasms primary in vesical diverticula which he collected from the literature. In 1935 the reports of Hardwick and Priestley,[23] Owre[24] and Allen[25] made a

total of 44 cases of neoplasms primary in diverticula which have been reported in the literature.

Fig. 1. Case III. The catheter was passed into the diverticulum which was thoroughly lavaged and then filled with skiodan. A filling defect was seen which was due to a tumor.

Fig. 2. Case V. Roentgenogram showing filling defect in a diverticulum of the bladder after the contrast media had been removed from the bladder itself. This is due to a tumor in the diverticulum.

Incidence. Neoplasms primary in bladder diverticula are of rather rare occurrence, according to reports in the literature. Kutzmann[26] in reviewing a series of 100 diverticula of the bladder found no evience of a tumor in any instance. Of the 133 cases reported by Judd and Scholl,[11] carcinoma was primary in a diverticulum in only 4. At the Cleveland Clinic we have seen 221 patients with diverticula of the bladder, only 5 of which contained primary tumors.

Age. A review of the literature reveals that neoplasms primary in urinary diverticula occur more frequently in adult life between the ages of forty-eight and seventy-six years.

Buerger, Blum, Lowsley and Gutteriez, and Rathbun have reported that the ages of their patients varied from forty-five to fifty years. In this series, the ages of the patients were seventy-four, sixty, sixty-nine, fifty-nine, and fifty-five years.

Symptoms. Hematuria is the outstanding symptom of this condition. It occurred in 83 per cent of the reported cases in the literature, and in all those reported here. In the case reported by Deming, hematuria had been present for three weeks while in that reported by Judd and Scholl, it had been present for three years.

All the patients in our group complained of symptoms of vesical neck obstruction, such as difficulty in voiding, hesitancy, dribbling, frequency, and nocturia. Although pain has been reported in some of the cases in the literature, it was strikingly absent in our patients. Retention of urine has been reported in many instances, and marked frequency and dysuria may occur when a coexisting infection is present.

Diagnosis. In the majority of patients, diverticula of the bladder may be diagnosed readily by cystoscopic and roentgen examinations. In the instances in which blood can be seen spurting from the orifice of the diverticulum, the presence of a neoplasm should be suspected. Likewise if a tumor protrudes from the diverticulum,

the diagnosis is readily established. However, the tumor may be small and located at the bottom of a diverticulum thus

Fig. 3. Intravenous urogram showing both kidneys to be normal. The right ureter empties into a diverticulum of the bladder which shows a constant filling defect.

preventing its visualization. In a small percentage of such cases, however, the cystoscope may be pushed into the diverticulum and the tumor visualized.

In 2 of our cases, after thorough lavage of the diverticula, persistent filling defects were noted upon two successive examinations and preoperative diagnoses were made which were verified at the time of operation. In one case, the tumor was visualized at cystoscopic examination.

In the 41 cases reviewed by Le Comte, a diagnosis was established in 21 patients at operation, at necropsy in 10, by cystoscopic examination in 9 and in one instance in which the cystoscope was introduced into the diverticulum, a tumor could be seen. Cystograms were made in 14 cases and they demonstrated filling defects in 6; however, cystograms were not employed if the tumor was visualized during cystoscopic investigation.

Rectal examination is extremely important. The prostate may or may not be enlarged but above the prostate, a hard,

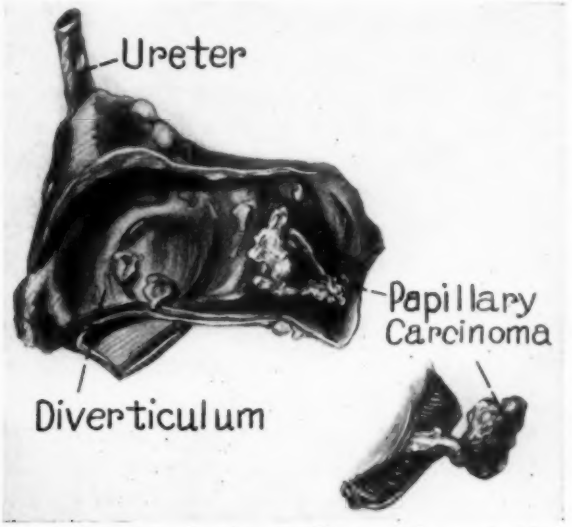

Fig. 4. Drawing showing papillary carcinoma in bladder diverticulum.

firm, fixed mass is sometimes palpable which may be responsible for an erroneous diagnosis of extensive carcinoma of the bladder or prostate unless the possibility of this condition is kept in mind.

A preoperative diagnosis of tumor confined to a diverticulum was made in 3 of our 5 cases, and these diagnoses were verified at operation.

Pathology. The neoplasms which occur in diverticula may be benign or malignant. A review of the cases reported by Le Comte[3] reveals the following types:

Sarcoma	2
Cavernous hemangioma	1
Epithelioma	1
Epidermoid carcinoma	2
Squamous cell carcinoma	5
Carcinoma	20
Papilloma	4
Benign papilloma	2
Malignant papilloma	1
Epithelial neoplasm	1
Polyps	2

Since Le Comte's review, one carcinoma[23] and two papillary carcinomas[24,25] have been reported. Two benign papillomas, one carcinoma and two papillary carcinomas comprised our series. Microscopic and gross examination showed that these tumors simulated those occurring primarily in the bladder.

Treatment. Total resection of the diverticula containing the tumor is the rational treatment. Whether the intravesical or the extravesical technique is utilized depends upon the preference of the surgeon. In this series, the extravesical technique was used, thereby completely excising the diverticulum with a portion of the bladder in the region of the orifice of the diverticulum. In 2 cases in which the ureters were contained in the wall of the diverticulum, it was deemed advisable to divide and ligate the ureters and not attempt to reimplant them into the bladder. Although fulguration of the tumor has been employed, radical operation seems preferable.

End Results. In the cases reviewed by Le Comte, cures by excision were reported in 13 cases; however, since seventeen months was the longest period of observation, the final end results cannot be evaluated. In the 5 cases reported here, one patient with papilloma of the bladder died of a cerebral embolism seventeen months after leaving the hospital but no recurrence had been experienced up to that time. One patient with carcinoma in a diverticulum died suddenly of pulmonary embolism on the twenty-first postoperative day. One patient with carcinoma in a diverticulum lived one year and six months without recurrence. Another patient died of an unknown cause two years after removal of a diverticulum containing a papilloma. The fifth patient is living without recurrence six months after operation.

CONCLUSIONS

Primary carcinoma of a diverticulum of the bladder occurs more frequently than we surmise. Complete investigation including cystoscopic examination and cystograms will frequently establish an accurate diagnosis before operation. Radical surgical excision is the procedure of choice.

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FRACTURE OF SPINE

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THE modern treatment of fractures of the spine is so radically different from that which was employed up to about ten years ago, that it may be assumed fairly that its aim, technique and results are not appreciated generally, particularly by the older general practitioners or those physicians not especially concerned with industrial or orthopedic surgery. It is, therefore, appropriate and advantageous to review this subject in some detail.

The former treatment of fractures of the spine depended on the following postulates:

1. The deformity of a fractured vertebra can not be corrected. Any attempt at correction was dispelled by fear of damaging the adjacent spinal cord.

2. A fractured vertebra heals very slowly, much more slowly than a fracture of any other bone.

3. Permanent disability is inevitable.

In the modern concept of the pathology and the prognosis of a compression fracture, none of these premises is tenable. The experiences and teachings of Arthur Davis of Erie, Pa., John Dunlop of Los Angeles, William Rogers of Boston, R. Watson-Jones of Liverpool and many others have brought to light facts which have completely altered our ideas in regard to the pathological anatomy and potential healing qualities of a fractured vertebra. To Arthur Davis belongs the credit for being the first to direct our attention from the old conservative methods to the opportunities presented by treating a fractured vertebra as one would any other fractured bone. In the management of any fracture we are governed by the principle that early reduction of the fragments and efficient immobilization will yield the best results. This principle is applicable in the treatment of fractures of the spine. We now know that: (1) It is possible to reduce the deformity and to return the fragments of a fractured vertebra to a normal or approximately normal relationship by appropriate manipulation; (2) the back can be immobilized so that the fragments of the injured vertebra are held in their proper position; and (3) a fractured vertebra with the fragments in good position will heal rapidly and thoroughly.

These are the factors which are guides in the care of a compression fracture of the spine and render the prognosis favorable. The graver aspects of a fracture of the spine do not depend on the injury to the vertebra but on the damage to the adjacent soft structures. This applies to the spine in no greater degree than to other bones. A fracture of a parietal bone has an excellent prognosis unless an underlying vessel has been ruptured or the cerebrum damaged. A fracture of the pelvis is not serious unless a pelvic organ or vessel has been torn. A simple fracture of the tibia may be very grave if the posterior tibial artery has been severed. Similarly, in a fracture of the spine the ultimate prognosis depends on the associated injury to the nerve roots and more particularly to the spinal cord.

PERTINENT ANATOMICAL FACTS

It is important to bear in mind the conformation and structure of the spinal column, and especially the architecture of the vertebra, in order to appreciate not only the mechanism and pathology of a spinal fracture, but more particularly those anatomical elements which facilitate the reduction of a fracture and provide support and protection to the injured area. The spine is a segmented column of separate bones, the vertebrae, joined by very strong intervertebral fibroelastic discs (Fig. 1) and numerous ligaments. Of the latter the

most important is the anterior common ligament (Fig. 1). This structure is a strong and tough sheet of fibrous tissue

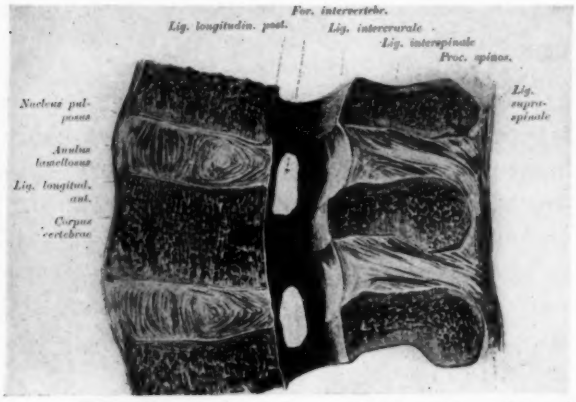

FIG. 1. This illustration of a vertical section through the lumbar vertebra is taken from Fick's Handbuch der Anatomie und Mechanick der Gelenke. Note in particular the strong anterior common ligament which binds the vertebral bodies. This illustration shows very clearly that the greatest part of the vertebral body consists of cancellous, very spongy bone surrounded and encased by a thin layer of cortical bone.

extending along the front and sides of the vertebrae, continuously from the upper cervical region to the sacrum. In the ordinary compression fracture this ligament is not damaged much and lies in folds clinging to the fragments at the site of the fracture. When the fragments are realined it becomes taut, and in hyperextension of the spine, as will be described presently, it acts as a splint to hold the fragments fixed in their proper position. Each vertebra consists of a body and a posterior arch from which project the spinous, articular and transverse processes. The body has a thin layer of cortical bone about the periphery which encases spongy, vascular cancellous bone. On the posterior surface of the bodies there is the posterior common ligament (Fig. 1) which extends along the entire length of the spine, and, though it is thin and less effective than the anterior common ligament, it affords some support to the vertebral fragments of a reduced fracture.

From a practical standpoint there are three types of fractures to consider in an injury of the spine: (1) compression fracture of the vertebral body without cord involvement; (2) fracture of the vertebral processes; and (3) fracture of the spine

FIG. 2. Shows typical wedging of a fractured vertebral body. The base of the wedge is posterior and the apex anterior. The adjacent intervertebral discs are undisturbed. The fracture line is through the substance of the body. The inferior surface is not altered and the superior surface is only slightly uneven.

with cord involvement.

COMPRESSION FRACTURE OF THE VERTEBRAL BODY WITHOUT CORD INVOLVEMENT

Etiology and Pathology. This is the commonest of the spinal fractures, usually resulting from an indirect violence, as a blow on the head or upper part of the back, or landing on the buttock or feet after a fall, in which act the spine is forced into acute flexion. The cancellous lamellae break, the body of the vertebra collapses, and the superior surface is approximated to the inferior. Usually there is greater pressure on the front than on the posterior part of the body so that the latter is weged shaped, narrow in front and high in the

back (Fig. 2). The superior surface of the body may remain intact, as when the upper part of the body is impacted into the lower, the fracture line extending through the middle of the body. The superior surface or plate may be fractured in one or more places. There may be several lines of fracture through the body, a comminuted fracture, with or without separation of the fragments. When the major force of the injury extends through the middle of the vertebral body the greatest collapse and compression will take place there. Occasionally the fracture occurs through forcible hyperextension of the back. In these rare instances the back of the body may be compressed more than the front and the wedge is reversed. Several vertebrae, either contiguous or with intervening uninjured vertebrae, may be fractured. The fracture of the body usually exists as an isolated lesion, but there may be an associated injury of one or more processes, with or without dislocation of the body (Fig. 8). The most common type, however, is a compression fracture of a single vertebra, the wedge having its base posteriorly, the fracture resulting from a sudden flexion of the spine. There may be a predisposing factor such as senile osteoporosis (Fig. 3), osteoporosis secondary to an endocrinopathy, or a metastatic malignant focus which has previously weakened the framework of the vertebra.

Frequency of Involvement of Different Sections of the Spine. From our knowledge of the structure of the vertebral column we might expect that the dorsal vertebrae, especially the first to the tenth, would be involved infrequently because of the protection of the adjacent ribs which at their vertebral extremities are connected to the vertebrae by numerous strong ligaments. This is substantiated by experience, for in any large group of cases of fracture of the spine the first to the ninth dorsal vertebrae, inclusive, are found to be involved the least often. The greatest number of fractures are found in the lumbar region including the tenth, eleventh and twelfth dorsal. The cervical vertebrae are affected next in frequency. Boorstein,[1] reviewing 41 cases gives the following figures:

	Cases
Cervical	15
Dorsal 1-11th	4
Dorsal and lumbar	4
Lumbar	18
Total	41

Cleary[4] reports the following distribution in 47 cases:

	Cases
Cervical	7
Dorsal 1-11th	9
Dorsal 12th	5
Lumbar	26
Total	47

I recently reviewed 98 cases of fracture of the spine treated at my office or admitted to the Hospital for Joint Diseases. The following indicates the sites and frequency of involvement.

	Cases
Cervical vertebra	17
Dorsal vertebra (1st to 9th)	5
Dorsal vertebra (10 to 12th)	18
Lumbar vertebra	37
Sacrum	1
Coccyx	1
Transverse processes	13
Spinous processes	4
Articular	1
Odontoid	1
Total	98

There were undoubtedly many unrecognized instances of fracture of the lamina, pedicles and articular processes, but their presence is often either obscured by the shadows of the bodies or overlooked through the greater interest in the injury to the bodies.

Symptomatology. The severity of the symptoms varies with the intensity of the injury. In the uncomplicated fracture the patient may, at the time of injury, experience only a sense of stiffness or lameness of the back and very little disability.

Case 1. R. S., a powerfully built young man, was in an automobile accident in which he was thrown to the ground sustaining a

fracture of the body of the second lumbar. He felt a slight discomfort in his back. So little, however, that on the following evening, not appreciating the nature of his injury, he engaged in a wrestling match in which he made a creditable showing.

Case II. Mrs. P. fell from a horse, fracturing her first lumbar. She had so little inconvenience that it was not until three weeks later that she sought medical advice.

The subjective symptoms are not commensurate with the physical alteration of the injured vertebra. Not infrequently there is loss of consciousness from concussion of the brain and collapse and shock resulting from damage to the abdominal or Thoracic organs, or fractures of the extremities, so that the *fracture of the spine may be overshadowed for several days or even several weeks by the injury to other parts.*

Case III. John P., a structural iron worker, was compressed between steel girders. He was brought to the hospital in marked shock with an increasing emphysema of his chest and dyspnea. It was not until several days later when his general condition improved that he complained of pain in the neck which was found to be due to fractures of several of the cervical vertebrae.

For purposes of clarity it will be convenient to study the clinical picture of an uncomplicated fracture without cord involvement.

SUBJECTIVE SYMPTOMS

Backache. The commonest complaint is pain in the back in the exact location of the injured vertebra. The area of the pain is comparatively small, as it extends very little beyond the limits of the vertebra. The pain is usually mild and only exceptionally severe. It is, however, *persistent*, is increased by any activity, and is eased only by rest or support of the back. The degree of the pain in the back depends in some measure on the location of the injury. It is least marked in the dorsal region where there is normally little movement and where the injured vertebra is protected by an extensive network of ligaments joining the vertebrae to one another and to the adjacent ribs. The pain is likely to be marked in the neck where there is free motion between the vertebrae. In the lumbar region the pain depends on the degree of fragmentation and the deformity.

Referred Pain. As the spinal nerves course through the intervertebral foramina they may be pressed upon, with the result that there is pain referred to their distribution. Thus an individual who has sustained a fracture of the spine may suffer pain in the upper extremities, chest, abdomen or the lower limbs depending on the level of the injury. Exceptionally the referred pain may be so severe as to overshadow all other complaints and lead to an erroneous diagnosis of a peripheral lesion or one in the chest or abdomen rather than in the spine, especially if there is uncertainty about the manner of injury or if the other symptoms in the back are mild.

Case IV. Mrs. M., sixty-two years of age, lifted a grandchild. She felt some little pain in the back but thought nothing of it. She soon began to have pain in the lower limbs. This persisted and many examinations of the hips and knees were made to discover its cause. This patient also had some stiffness of the back but this did not attract so much attention as the discomfort in the lower limbs. It was nearly three weeks after the original injury before it was discovered that she had a fracture of her first lumbar vertebra (Fig. 3).

Referred pains are likely to be common when the intervertebral canals have been reduced in size, as through extensively comminuted fractures or fracture-dislocations of the vertebral body, fractures of the lamina or pedicles, associated with the spinal canal, displacement of fragments or an exuberant callus, which occurs rarely, formed during healing of the fracture.

Weakness of the Back. Almost immediately after the injury and continuously thereafter the patient feels a weakness of the back. He is unable to lift objects or to sit or stand in one position for any length of time. He is constantly seeking to rest

and feels better when lying down than in any other position. The weakness may persist for months, years or even permanently. The persistence of this symptom is likely to be found in those cases in which the fracture was not reduced or the deformity only partially corrected.

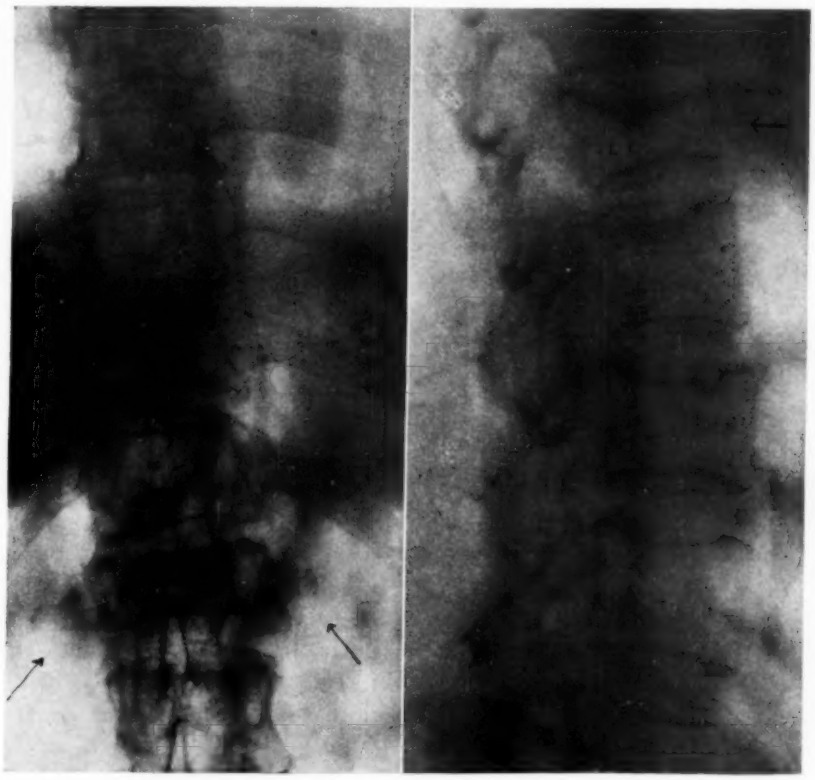

Fig. 3A. Fig. 3B.

Fig. 3. Compression fracture of L-1 secondary to senile osteoporosis. A. Anteroposterior view. Note rarefaction of all of the vertebrae. This is very evident in the original film. The vertical diameter of L-1 is greatly reduced. There is some increase in the transverse diameter. The intervertebral discs are intact. B. Lateral view. The general osteoporosis is here apparent. Note the wedging of the body of L-1 and no involvement of the adjacent discs.

Stiffness of the Back. The patient cannot bend forward, backward or sideways as freely previously. The stiffness is not equally marked, but it is present in all directions. The degree of stiffness varies with the extent of the fracture, the acuteness of the backache and with the amount of activity. When the patient first arises from a rest it may be marked. After a little activity the sense of stiffness diminishes, but it increases decidedly if the individual continues to stand or walk for several hours.

Weakness of Extremities. As a result of pressure on the peripheral nerves there may ensue a variable degree of weakness of the muscles of the extremities, up to total paralysis of one or more groups. This is seen particularly frequently in fractures or fracture-dislocations in the cervical region (Fig. 12). Not only has the patient pain in the arm but inability to raise it because of weakness of the shoulder muscles or of the hand and wrist muscles. Should there be pressure upon or destruction of the cord the symptoms will vary from temporary motor weakness to total permanent paralysis with loss of sensation and control of the rectal and vesical sphincters.

Deformity of the Spine. Only exceptionally is the patient aware of a deformity of the spine. The size of the "knuckle" is comparatively small and, even though by chance the individual touches it, he is not likely to appreciate its presence. It certainly is rare for the patient to complain of a deformity.

OBJECTIVE SYMPTOMS

Localized Tenderness. Just as localized pain in the spine is the commonest of the subjective symptoms, so localized tenderness at the site of the backache is the most constant and most significant of the objective findings. The tenderness is definitely limited to the injured vertebra and by its localization indicates the vertebra involved. It is important to appreciate that in a fractured vertebra the tenderness is found only on pressure upon its spinous process and is absent elsewhere, as in the musculature on either side. The tenderness is of a mild character and never acute as in an inflammatory process. It is, however, persistent and may be elicited for several months after the original injury.

Deformity. With a compression of the injured vertebra there is a certain amount of buckling and angulation of the spine so that the spinous process of the injured vertebra is abnormally prominent and projects backward beyond those on either side causing a "knuckle." The degree of the deformity varies. When marked it is visible in all positions of the trunk. When mild it may become apparent only when the patient bends forward. It is easily overlooked if slight, especially if the patient is stout or muscular. But, though it may not be visible, it is always palpable. When one runs his finger down the spine of a normal back he senses that the curve of the line of the spinous processes, that is the physiological anteroposterior curve of the spine, is unbroken. But in a spine with a fractured vertebra this line is broken at the level of the fracture, the examining finger feeling the abnormal backward projection of the spinous process. This break in the physiological curve of the spine corresponds to the area of local pain and tenderness, and is an important objective sign. Frequently in the cervical and lumbar regions the angulation at the site of the fracture causes a marked flattening or even reversal of the normal forward curvature.

Limitation of Spinal Motions. If the fracture is in the dorsal region the motions of the spine are restricted but little, in the cervical and lumbar areas the limitation of motion is definite and easily recognizable. All the motions are limited, though not equally in all directions. The fact that all of the motions are restricted indicates that the spinal column and not the adjacent soft structures is involved.

Muscle Spasm. There is usually evident a moderate degree of spasm of the erector spinae muscles in the area of fracture and for some little distance above and below it. This serves to splint the spine and restrict movement which would be painful. The muscle spasm is most marked in the neck where there is normally very free motion, and least in the dorsal region. In fact in a mild fracture of a middorsal vertebra the free spinal movements may be misleading and lead to a dismissal of the consideration of the existence of a fracture.

Disability. As a result of the pain, stiffness and muscle spasm there is a disability which includes awkwardness in walking, difficulty during changes in position, inability to lift objects, and in some cases actual paralysis.

Sensitiveness of Back from Indirect Pressure. An important but frequently neglected finding is pain in the injured part of the spine on indirect pressure, such as tapping the patient on the head or having him jump down on his heels. This is not peculiar to fractures, but indicates a painful lesion and is of value only in conjunction with other symptoms.

X-ray Examination. Although the history of the injury and the symptoms can with reasonable assurance establish a diagnosis of a fracture of the body of a vertebra, positive proof is obtainable only through a roentgenogram. Two views are advisable, an anteroposterior and a lateral. Anteroposterior view (Fig. 3): In this picture several findings of importance are seen. The most frequent change is a reduction in the vertical diameter of the vertebra. This varies in degree, but even in the

mild cases is definite. The texture of the bone appears unaltered, only the size is reduced. When the fracture is comminuted, displaced fragments may be seen projecting across the adjacent intervertebral spaces or more commonly laterally on one or both sides. The compression may not be uniform or symmetrical, hence the body may appear wedge shaped. In the fracture-dislocations the body is displaced laterally, and forward, commonly in the cervical area, or backward, in the dorsolumbar region (Fig. 8). It is sometimes dislocated laterally to a surprising extent without associated cord involvement. Lateral view: This picture is the one which contributes the diagnostic feature (Fig. 2). The characteristic appearance is wedging of the vertebral body, the anterior border being short and the posterior border the base of the wedge. In such wedging the lower surface remains undisturbed while the upper surface is oblique as if the wedging or crushing took place from above downward. Both the superior and inferior surfaces may be smooth, the actual fracture line being more or less transverse through the substance of the body. The fracture line or lines may extend through the superior plate as well as through the body of the vertebra, exhibiting distinct solution in the continuity of the cortical bone of the superior and other surfaces of the body (Fig. 3B). When the crushing force passes through the middle of the body this portion is reduced in its vertical diameter while the anterior and posterior borders are altered but little. Cases are recovered, though I have not seen any examples, in which the posterior part of the body has been crushed through a force following severe hyperextension of the spine. In such instances the base of the wedge is situated anteriorly. In comminuted fractures fragments of bone may extend in any direction, but particularly upward and forward. If only one vertebra has been broken the intravertebral disc will not be altered. But when there is an extensively comminuted fracture, or more particularly when several adjacent vertebrae are fractured, or in a fracture-dislocation, the disc or discs may be severely disrupted and the areas occupied by them reduced in size or obliterated.

SUMMARY OF SYMPTOM COMPLEX INDICATIVE OF FRACTURE OF SPINE WITHOUT CORD INVOLVEMENT

The facts which indicate the existence of a fracture of the spine are briefly the following:

1. History of an injury in which the trunk was forcibly flexed through a direct or indirect injury or in which the muscles of the trunk suddenly and violently contracted when the body was in flexion.

2. Subjective symptoms of definitely localized pain in the back, weakness and stiffness of the back, referred pains in the extremities, chest or abdomen and weakness of one or more limbs.

3. Objective findings of localized tenderness, deformity of the spine, awkward gait, restricted spinal motions, spasm in the back muscles, paresis or paralysis and *x*-ray evidence indicating the exact location, type and extent of the fracture.

FRACTURE OF THE VERTEBRAL PROCESSES

Any of the vertebral processes may be injured, but the transverse processes are more commonly broken than the others.

Fracture of the Transverse Processes. Fractures of the transverse processes are rather common; in a recent review of 98 cases of fractures of the spine at the Hospital for Joint Diseases there were 13 instances of fracture of transverse processes, all in the lumbar vertebrae. One or several processes may be broken (Fig. 4), but I have seen only those of the lumbar vertebrae involved. This seems natural as those of the dorsal vertebrae are thoroughly protected by the overlying ribs. In the cervical vertebrae the transverse processes are uncommonly strong, so that presumably a traumatic force applied to the neck is transmitted to the bodies, discs or intervertebral articulations. The fractures of the lumbar transverse processes are

almost always a simple linear break, usually vertical or slightly oblique only very rarely transverse and may be through any part of the process. In a small minority it is comminuted. The fragments may not be separated by more than the fracture line or the outer fragment may be displaced upward or outward for a considerable distance. The etiological force is a direct blow, though instances have been seen in which a strong muscular effort was the immediate cause.

Fig. 4. Unilateral fracture of transverse processes of L-3 and 4. Note the marked separation of the fragments.

Symptoms. The symptomatology of fractured transverse processes is very vague. There may not be any symptoms whatever, not unusually there is indefinite pain over a wide area due manifestly to associated soft tissue injury. The tenderness is generally limited to the fracture. Those motions of the spine which cause tension on the injured area are limited and painful. The diagnosis can be made only by finding the fracture in a roentgen ray picture. In the ordinary civilian a fractured transverse process causes little or no disability. The fracture heals promptly or if the fragments are separated there is a brief period of discomfort and disability and the situation is forgotten. In medico-legal and compensation cases who have been told that they have a fracture of the "back" the symptoms persist indefinitely until a financial settlement has been agreed. Where there is no legal obligation I have never seen a fracture of a transverse process give extended disability.

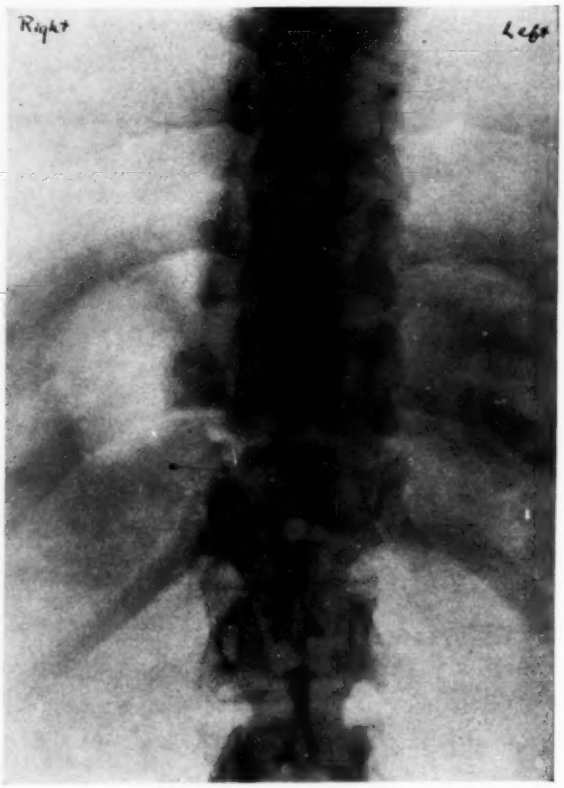

Fig. 5. Fracture of right lamina of D-12.

Fracture of Spinous and Articular Processes and Pedicles

These elements of the vertebra are, in comparison with fractures of the body or even the transverse processes, very rarely fractured. The cause is a direct blow. The fracture is usually linear with little or no displacement (Figs. 5, 6 and 7), although occasionally there are multiple linear

fractures with no separation of the fragments or very slight displacement. This type of fracture may be isolated or more commonly a part of a more extensive injury to adjacent bones or soft tissue. The symptoms consist of pain in the region of the injury, increased by activity, especially movements of the spine, and relieved by rest. It is particularly noteworthy that the pain is increased only by certain motions of the spine, namely those causing tension on the fractured area. There is local tenderness but no deformity. Walking and general activity are impeded to a variable degree depending on the individual sensitiveness. A positive diagnosis is possible only by roentgenographic evidence of the fracture. The films must be carefully inspected in good light, otherwise the fracture line or lines may be overlooked.

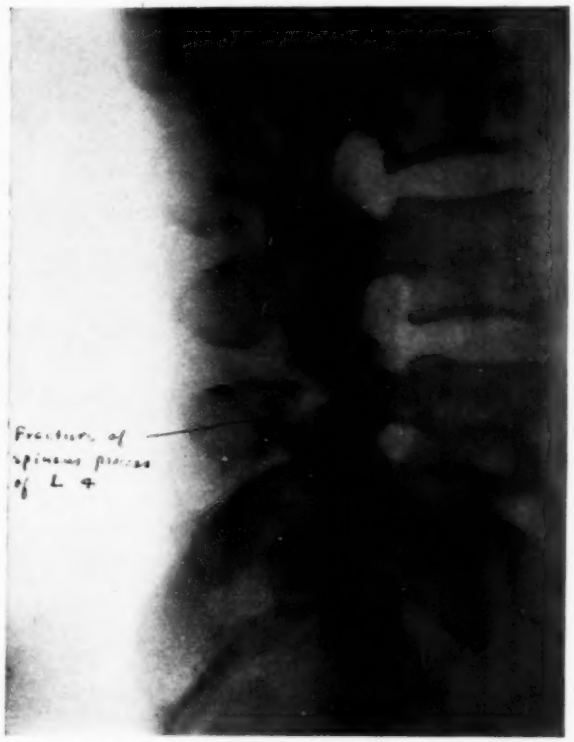

Fig. 6. Vertical fracture through base of spinous process of L-4 with moderate separation of the fragments.

FRACTURE OF THE SPINE WITH CORD INVOLVEMENT

In a fracture or fracture-dislocation of a vertebra the spinal cord may be crushed or severed with complete, irreparable paralysis below the level of the injury (Figs. 8 and 9). The cord may, however, only be compressed by a displaced fragment of bone, hemorrhage or edema. Pressure in the latter conditions may cause permanent damage if continued long enough. Several practical problems face the surgeon in this emergency. If there is a recent fracture or fracture-dislocation in the neck with partial paralysis of the upper limbs, reduction of the fracture or fracture-dislocation should be undertaken promptly with the confidence that when the fragments are realined the paresis or paralysis will disappear (Fig. 10). If the injury is ancient it is doubtful how much improvement and recovery can be obtained. If the osseous lesion warrants it, a reduction of the deformity may be attempted but with considerable reservations as to the outcome. If a reduction is not feasible or permissable because of the age of the lesion or its location, one must reconcile himself to the existing disability, although even in such cases support of the neck may give some relief. So far as the limb is concerned, if there is only partial disability, some supportive or operative measures applied to the limb may yield improvement (Fig. 11).

Fig. 7. Fracture through base of odontoid process.

A difficult situation arises in relation to the procedure to be followed in recent injuries with complete paralysis. Some believe that an immediate laminectomy is indicated; others are vigorously opposed to

it. If the paralysis is due to a crushing or severance of the cord, surgery is useless, but if a fragment of bone is pressing on the cord, its early removal and the release of the cord are desirable; if the nerve symptoms are due to hemorrhage into or edema of the cord, rest will suffice to relieve them. What shall one do in a given case with total paralysis? If the *x*-ray pictures give indubitable evidence of the displacement of a fragment and pressure by it on the cord then resort to a laminectomy should be prompt. If there is a dislocation of the body of the vertebra (Fig. 8) so that a severance of the cord has undoubtedly occurred, it serves no purpose to operate. If there is no gross dislocation of the vertebral body and if there is no proof that a fragment of bone is pressing on the cord, and it is likely that the injury has given rise to hemorrhage and edema of the cord, the patient ought to be kept very quietly in bed and given an opportunity for spontaneous recovery which may and often has taken place.

Fig. 8. Fracture dislocation of D-12. Note the marked compression and wedging of the body of D-12 and the marked backward dislocation of this vertebra amounting to a full half inch. The intervertebral discs have not been injured as is seen in the film taken after the reduction (Fig. 9).

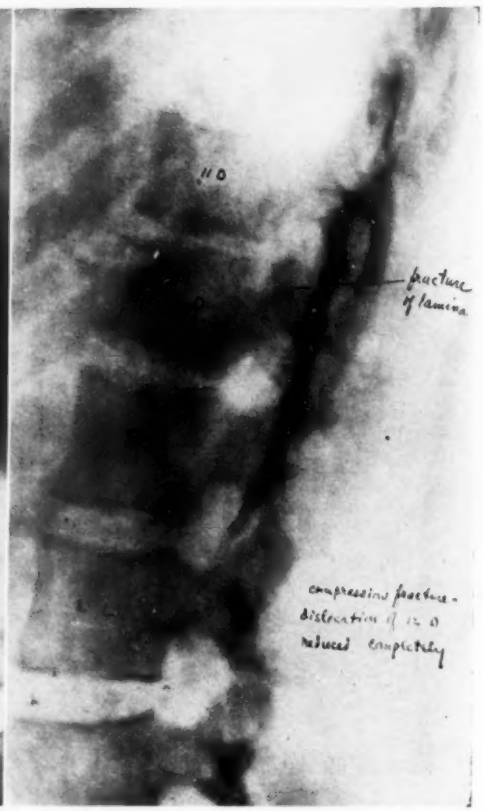

Fig. 9. Same case as in Fig. 8. Lateral view taken after manipulative reduction by the Watson-Jones method. Note complete restitution of the form of the body of D-12 and complete reduction of the dislocation.

These criteria are not comprehensive nor entirely satisfactory, but they are valuable from a practical point of view, considering our severe limitations. Laminectomy is a comparatively simple operation, yet in fractures of the spine, performed routinely for paralysis, it is attended by more than average risk. Years ago this operation had a high mortality rate, but even today our inclination to decompress the spinal cord must be tempered with the knowledge of the following:

1. If the pressure has existed for a long time the existing damage is permanent.

2. Paresis from hemorrhage or edema subsides when the blood or exuded fluid is absorbed.

3. If the injury is such that it is likely that the cord is severed, laminectomy adds an operative insult to an existing severe injury. The futility of this operation becomes painfully apparent after one has done the operation and found a completely crushed cord.

Fig. 10A.

Fig. 10B.

Fig. 10. A. Fracture dislocation of C-4. This patient had pain and weakness of arms. These symptoms were still present when first seen several weeks after the injury. He had worn support to his neck ever since the injury. B. (Courtesy of Dr. Menzies.) The deformity had been reduced by applying traction and a plaster of Paris collar. The reduction could not be maintained and hence resort to a "distraction collar" was necessary. This gave ample support and relief from discomfort in his neck and arms. Nine months after the injury, he has about 75 per cent of normal motion in the neck, no tenderness or pain in the neck and has returned to his former work which is that of a fitter in a men's clothing store.

In the face of a total paralysis following a fracture or fracture-dislocation of the spine an immediate reduction would be attempted. If after the reduction the paralysis persisted I would do a laminectomy, providing there is convincing evidence of pressure on the cord from a fragment of bone. If there is no such proof the patient should be kept at rest on a convex frame and hope that the paralysis is the result of hemorrhage into or edema of the cord, and that when the extravasated fluid has been absorbed the nerve symptoms will disappear or be ameliorated

TREATMENT

Compression Fracture of the Vertebral Body without Cord Pressure. The essential principle involved is, as in any fracture, reduction by realinement of the fragments. The problem is simplified by the fact that realinement is readily accomplished by hyperextension of the spine, which is maintained until healing occurs. Hyperextension of the spine can be obtained through any of several techniques. Davis[7] describes his method as follows:

The anaesthetized patient is laid prone on a low table. The usual head suspension apparatus is used to completely elevate the lower extremities until the pelvis clears the table by several inches. The suspension is done in a direction upward and footward. That is, the center of suspension is placed about twelve inches distal to the foot, so that the traction is horizontal as well as vertical, the knees flexed at 45° rather than 90°, thus obtaining horizontal traction as well as hyperextension, also minimizing the pressure on the posterior arch of the involved vertebra. After sufficient suspension has been gained, manipulation is made by way of several quick but measured downward thrusts at the site of the gibbus. In many cases this manipulation is unnecessary for the reason

that the knuckle disappears with suspension. It is only in those cases where the knuckle still remains prominent that the manipulation is necessary to disengage the impaction, reduce dislocation or correct alinement. A posterior padded plaster shell is then made from the head to knees. The roller is mounted and lashed on with plaster ropes. Draw sheets, which previously have been placed transversely under the patient are now drawn tightly around the shell, and the complete ensemble is then rolled into the dorsal position. The anterior shell and rollers are then made. The life preserver roller was selected because of its lightness, firmness, adaptability and availability. Many materials might do as well.

Fig. 11. Distraction brace for support of the neck in a fracture of a cervical vertebra.

Dr. William A. Rogers devised a hyperextension frame which is an adaptation of the convex frame. It is arranged so that it can be gradually made convex to almost 90°. The patient lies on this frame and as the convexity is increased a marked and even extreme degree of hyperextension of the spine is obtained. The patient then lies in this position until healing takes place.

Dr. John Dunlop uses an apparatus like the Goldthwaite frame with an automobile jack to obtain hyperextension. He then applies a plaster of Paris jacket.

My preference is for the method described by Mr. R. Watson-Jones of Liverpool. He places the patient in the prone position between two tables of different levels, one being about a foot higher than the other (Figs. 12 and 13) and no anesthetic is required. The arms and shoulder girdle rest on the higher table and the pelvis and lower limbs on the lower table, with the body sagging into hyperextension between them. The longer the patient lies in this position the greater becomes the degree of hyperextension. This can be increased by manual pressure on the fractured vertebra. A stockinette having previously been applied to the trunk, as soon as the limit of hyperextension is reached the bony prominences, as the sacrum, iliac crests, etc., are covered with felt and a plaster of Paris jacket is applied extending anteriorly from the symphysis pubis to the upper border of the sternum, and posteriorly from the upper dorsal region to well over the buttocks. The patient is kept in bed from one to several weeks with two weeks as an average, and then is allowed up for increasing periods each day. The original plaster jacket is retained for two months, then it is replaced by a spinal brace to be used for three to six months, depending on the rapidity and solidity of the union as determined by the patient's feelings, the objective symptoms and the roentgen-ray appearance.

The preceding type of plaster jacket is used in cases of fracture from the middorsal

to the lower lumbar areas. When the cervical or upper dorsal vertebras are fractured, the plaster support must be extended upward to include the head. In the cervical region a plaster of Paris collar or brace is a very convenient support, the reduction being best accomplished by manual traction on the head or traction through a flannel halter.

The most favorable time for reduction in a fracture of the spine is immediately after the fracture has occurred. Practically, a good result can be obtained within the first two weeks. I have a splendid result in a woman who came to me three weeks after a fall from a horse. During the first two weeks after a fracture of a vertebra little union takes place, so that the fragments can be readily separated and realined in a normal manner. However, after three or more weeks enough callus has been deposited to interfere with reduction.

MECHANISM OF REDUCTION

As is well known the intervertebral fibrocartilaginous discs, very strong and tough structures, are so resistant that they are not damaged much, if at all, by a fracture of a vertebra. The superior and inferior vertebral surfaces remain attached to the discs. As the spine is hyperextended the adjacent discs on either side of the fractured vertebra are separated carrying with them into opposite directions the fractured fragments of the vertebra. The separation of the collapsed or impacted bony fragments is controlled by the range of hyperextension, that is, the fractured upper and lower vertebral fragments are separated to a degree and position which is normal for them in hyperextension, consequently to the normal extent. The rearrangement of the fragments into their normal position is further aided by the strong limiting anterior and posterior common longitudinal ligaments or membranes. In addition the tension of the long anterior and posterior vertebral common ligament serves to hold the fragments in place in hyperextension during the process of healing. Thus the vertebral body, which can be considered as a box supported by the intervertebral discs above and below it and by the common longitudinal ligaments in front and in back, is reestablished in both size and form.

In the neck the fracture is usually accompanied by some degree of dislocation, which is usually greater than the fracture. The longitudinal traction serves to realine the vertebral bodies completely or partially. Often one succeeds in only partially reducing the dislocation (Fig. 10). It is important to know that in partial reductions in the neck the result may be eminently successful so that the use of very great force is not necessary. A moderately ambitious effort should be made to reduce the dislocation, but should one obtain only a partial reduction, one need not subject the patient to extraordinary violence as the clinical and functional result will, in the majority of cases, be satisfactory.

DURATION OF SUPPORT AFTER REDUCTION

In the dorsal and lumbar regions six months after the reduction, and sometimes sooner, the fracture lines are not visible, the texture of the vertebra is homogenous, the fracture appears healed, and the vertebra can be subjected to ordinary normal strain, so that external support may be discarded. But if there is any pain, weakness of the back or local tenderness the brace should be worn somewhat longer. Many who have treated fractures of the spine by the older methods are now surprised at how complete the healing can be in six months after treatment is instituted.

In the cervical region, especially if the reduction is incomplete, the support should be continued for at least a year to allow ample time not only for bony healing but for healing of the injured soft structures which, presumably because of the mobility in the neck, remain sensitive a long time.

PROGNOSIS IN FRACTURE OF THE SPINE

The prognosis in fractures of the spine at the dorsolumbar junction, where so

many occur, is today vastly more favorable than in former years because of the advance in the method of treatment. Given a case

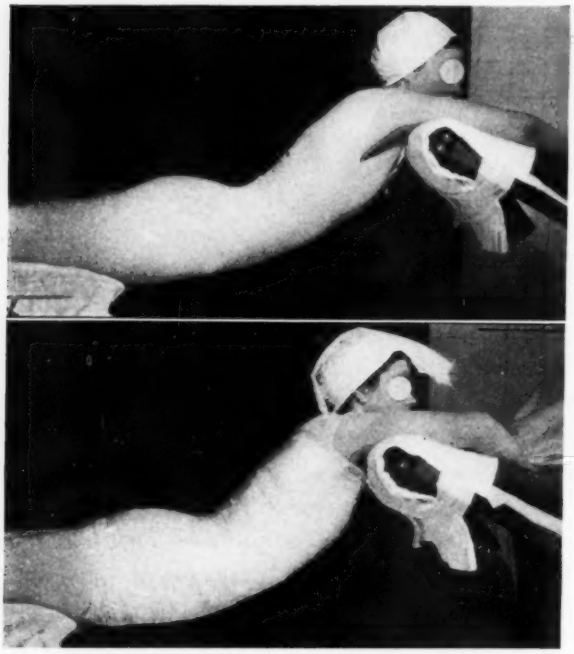

FIG. 12. The Watson-Jones method of reducing a compression fracture of dorsal or lumbar vertebra. The upper photograph shows the patient lying between two tables, the trunk sagging into hyperextension. The lower view shows the plaster jacket applied, the patient still resting between the tables.

soon after the accident and without nerve involvement, we can actually look forward to a complete restitution to normal and a cure in the majority of cases. In the last few years I have obtained a cure in every case of this type, most of the patients returning to their former habits of work and living. In the neck there is usually a partial disability because of either incomplete reduction, damage to the ligamentous structures, or both.

OPERATIVE FUSION OF THE SPINE

Has the procedure of fusion of the spine any place in the treatment of vertebral body fractures? Formerly this was commonly employed as a procedure of election in order to rapidly consolidate the vertebras in the area of the injury for the purpose of limiting pain and enabling the patient, especially if he is a laborer, to return to some gainful occupation. In an article in 1922[13] I summed up the consensus of opinion as follows:

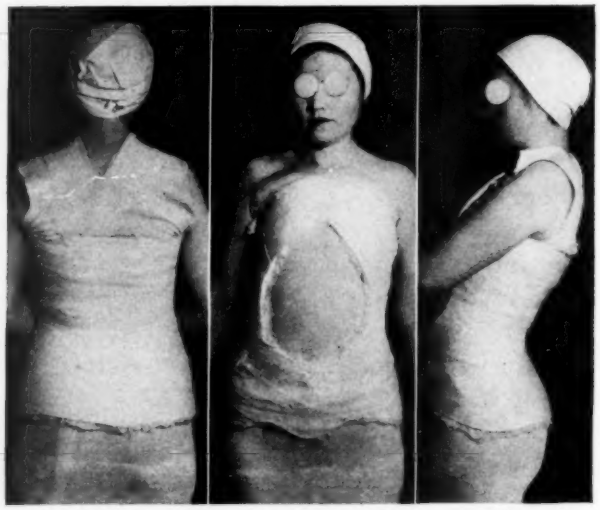

FIG. 13. Plaster of Paris jacket for fracture of the spine according to the Watson-Jones technique. Patient out of bed one week after jacket was applied. Note the attitude of hyperextension.

> . . . when shortening of the period of convalescence is indicated, then the radical or operative treatment is employed. . . . The operative treatment, through its evident advantages, would seem to be the treatment of choice. If the fractured spines were operated upon, let us say, within a month after the accident, we should, I believe, find rapid and early healing of the fracture, a great saving in time and reduction of the disability period.

Such a position is no longer tenable as our opportunities today are vastly better than they were then, but I believe that now the operative fusion of the spine for a fracture should be used as a last resort and not primarily, warranted only in (1) old untreated fractures of the dorsal and lumbar vertebrae in which there is marked and increasing deformity with backache and continuing disability; and (2) in irreducible or recurrent fracture-dislocations in the cervical vertebrae with pain and weakness in the neck and arms.

TREATMENT OF FRACTURE OF A VERTEBRAL PROCESS

In the case of a patient with a fracture of one or more of the vertebral processes

immobilization of the trunk by a plaster jacket or brace should be continued until the fracture is shown by *x*-ray films to be healed, or, if there is separation of the fragments with no opportunity for healing, until the patient is symptom free. In rare cases when a separated fragment of a transverse process continues to give backache and disability it should be removed. Many patients with fractured processes, especially transverse processes, have no symptoms referable to them and require no treatment.

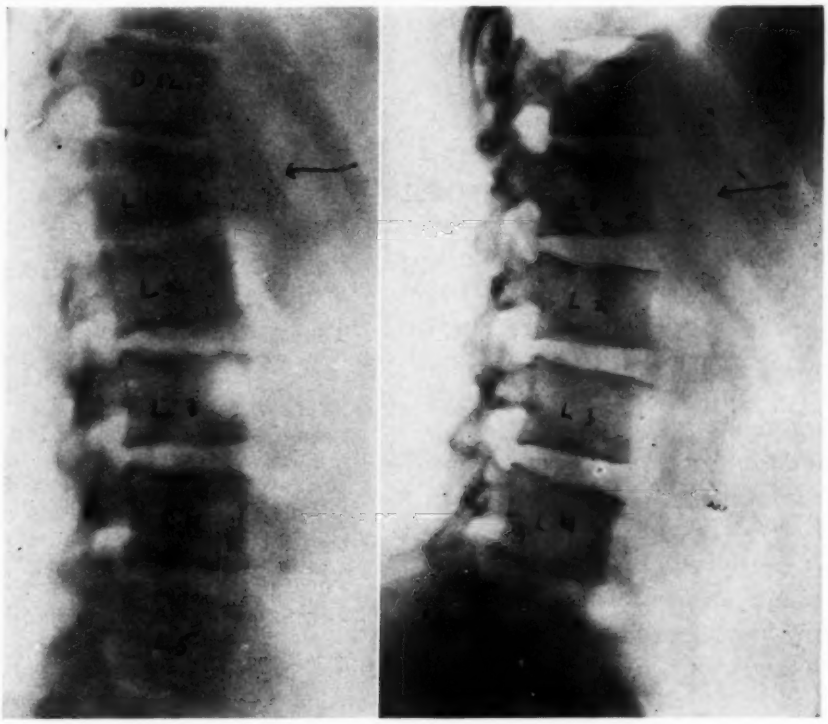

Fig. 14A. Fig. 14B.

Fig. 14. A. Compression fracture L-1 before reduction. Moderate wedging of injured vertebra and fracture through substance of vertebral body with little disturbance of superior surface. B. Compression fracture of L-1 after reduction, deformity almost completely corrected; solid bony healing. Mobility of spine reestablished.

Theoretically and legally a person with a fracture of a vertebral process may be said to have a fracture of the spine or back, but the damaging effect of such information on the morale of the patient is infinitely greater than the physical injury. The conscientious surgeon would, under such circumstances, avoid telling all of the truth to the patient. I have known several patients whose mental equilibrium has been seriously disturbed for it requires no stretch of the imagination to appreciate what such a statement would do to the sensitive and susceptible individual. In one instance the information was used for monetary returns, but the upset of the patient and the damage to his earning capacity was incalculable. I mention this point because in recent years there has crept into the profession a type of doctor who thrives on exaggeration of medico-legal cases, to the delight of the greedy lawyer.

TREATMENT OF A FRACTURE OF THE SPINE WITH PARALYSIS OF THE LOWER LIMBS

The treatment in this type of case may be divided into the early and late care. In the early stage the fracture should be reduced and immobilization continued until the bone has healed. The late care consists of nursing attention to prevent or heal pressure sores, and to prevent or delay an ascending infection of the bladder and kidneys. At best such patients are chair ridden, but with unusual care may be kept tolerably comfortable until the inevitable urinary infection and exitus.

CONCLUSIONS

The great advance in the treatment of fractures of the spine can only be appreciated when it is realized that the majority of the fractures are of the so-called compression type, and that the fracture involves most frequently the lower dorsal and the lumbar vertebrae, to which may be applied the remarkably effective hyperextension method. Formerly there was no thought of correcting the deformity, while today we can obtain a reformation of the shape and size of the injured vertebra. Formerly a patient with a fractured spine was doomed to permanent stiffness, disability and backache. Nowadays we look forward to not only anatomical restitution but to a functional cure. Operative fusion of the spine used to be considered a treatment of necessity, whereas now it is relegated to some ancient and untreated cases with persistent backache. Thus the prognosis in a compression fracture of the dorsal and lumbar vertebras is decidedly favorable. The treatment is simple, consisting of immobilization of the spine in hyperextension. It is further noteworthy that we now have the convincing proof that a fractured vertebra after reduction heals rapidly and completely.

The management of a fracture or fracture-dislocation of a cervical vertebra consists in the reduction of the fracture by hyperextension and traction and the immobilization of the neck and head in the best position obtainable until healing takes place. Complete reduction of a dislocation is not essential so long as there are no nerve symptoms. In the exceptional patient in whom the dislocation cannot be controlled by conservative measures, a spine fusion is indicated.

Fractures of any of the vertebral processes can simply and effectively be managed by immobilization of the back in a plaster jacket or spinal brace. In those cases in which there is no backache, treatment is uncalled. All the fractures in which the fragments are in contact and this includes the majority, heal rapidly. In an occasional case in which there is separation of the fragments and persistent pain, it may become advisable to remove the loose fragment.

Fractures of the spine with cord involvement should be treated conservatively and expectantly except when there is undoubted evidence of pressure of a fragment of bone on nerve tissue, when a laminectomy should be performed.

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CASE REPORTS

AUTHOR'S METHOD FOR REPAIR OF ANKYLOSED JOINT OF HAND

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CHICAGO

FOR some time we have been trying to arrive at a surgical measure for repair or rather, surgical reconstruction of an ankylosed joint in the hand. Prefacing this subject I do not think, although it may be possible, to apply this technique to fingers below the metacarpophalangeal joints.

This paper is written after having performed this operation on a number of cases, and I am showing the technique and end results on the last case. Unfortunately the previous cases operated, failed to report for final analysis.

This case was referred on February 5, 1935, with a history that in October, 1934, during an altercation, in striking he received lacerations over the metacarpophalangeal joint of the second finger on the dorsal surface, which became immediately infected and resulted in a complete ankylosis of this joint with the finger in complete extension but no loss of motion of the middle and distal interphalangeal joints (Fig. 1).

It was frankly explained to him that I thought I could help him but would promise nothing definite as to final end results. He assumed the responsibility of the operation.

I wish to lay considerable strength and emphasis on the fact that on these operations, the operator should not undertake any responsibility of operating on such a case until infection has disappeared and the tissues have remained healed for at least six months or longer.

On March 5, 1935, he entered the hospital and the affected hand and left leg were prepared by shaving the members, washing with green soap and water, cleansing with ether and applying an alcohol dressing, keeping an alcohol dressing on for twelve hours previous to the operation and then attempting to surgically

FIG. 1. Showing ankylosis of metacarpophalangeal joint.

execute, as nearly as possible, the Lane technique.

Under general anesthesia, a lateral incision was made on the dorsal surface laterally, and this incision can be made either on the thumb side or the ulnar side of the metacarpophalangeal joint (Figs. 2*a* and *b*).

The dissection is done very carefully, avoiding the sheath of the extensor tendon and drawing these structures to one side to expose the joint. The joint now being exposed and found to be ankylosed, a new joint is mapped out (Fig. 3). Too much emphasis can not be laid on this point; on insisting on the operator making a resection of the bone of the metacarpal at least ½ inch back of the original articulation, in order to give plenty of room for subsequent contraction of tissues following healing and in order to interpose the flap forming this new joint.

After this field is properly resected the whole joint area may appear quite loose and im-

FIG. 2a. Location of skin incision.

FIG. 2b. Healed wound following operation. Note some shortening of the operated finger.

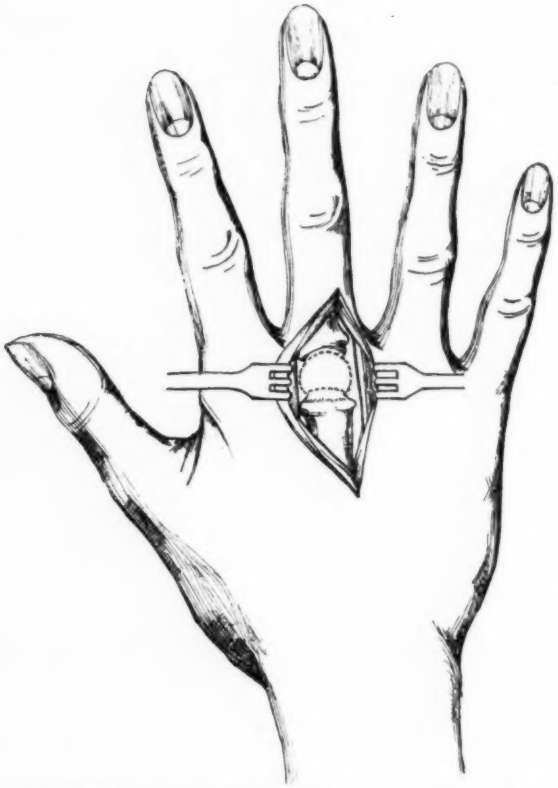

FIG. 3. Incision retracted, including extensor tendon; head of bone exposed for excision.

FIG. 4. Bone excised. Circular piece of fascia in place with circular string of catgut.

practical. However, this impression is changed as will be seen later.

The joint is now exposed and prepared for the plastic operation. From the thigh, an incision having been made lengthwise, the fascia lata is exposed and a piece removed from the surface sufficiently large to cover the distal end of the metacarpal (Fig. 4). The size of this fascia is best judged by a visual estimation of the appearance of the end of the metacarpal bone and then doubling it about 50 per cent upon removing the tissue.

I found it is better to have too much tissue to cover the end than not enough. Also, it was my experience, in cutting this fascia in a circular form from the thigh, that it was found very unsatisfactory and difficult to handle; therefore, this flap is removed somewhat in the form of a square and the edges trimmed off after the fascia is formed and tied around the end.

In using this fascia I have come to a very definite conclusion, the outer side of the fascia removed from the thigh should not be applied against the bone but just the reverse; the inner side should be applied, leaving the outer side exposed against the opposing phalanx and in this way the fat substance develops a normal lubricating surface. When this is accomplished properly nature cooperates. In fixing the fascial flap over the distal end of the metacarpal bone, a simple type of loop of plain catgut is sufficient (Fig. 4) and then the ragged edges beyond the circular or purse string can be removed.

It is essential, prior to applying the fascia, to control all bleeding by the use of bone wax; otherwise the fascial flap will be pushed away from the contact surface of the new made joint surface. The tissues are closed by ordinary method, using exceedingly fine catgut where necessary, preferably No. 00 pyoktanin or plain catgut. After the tissues are closed a simple fluffy dressing is applied without any fixation. This dressing is allowed to remain on for the first week without changing, assuming that the wound is clean. No unnecessary inspection should be made during this time in order to give nature a chance to heal the surgical field before postoperative education is begun.

Sutures are removed at the end of a week or ten days, at the same time re-education of the operative field is started by just a little stimulation of voluntary flexion and extension on the part of the patient. The re-education treatment consists of the cooperation of the patient in the flexing and extending of the finger, together with gradual active and passive massage.

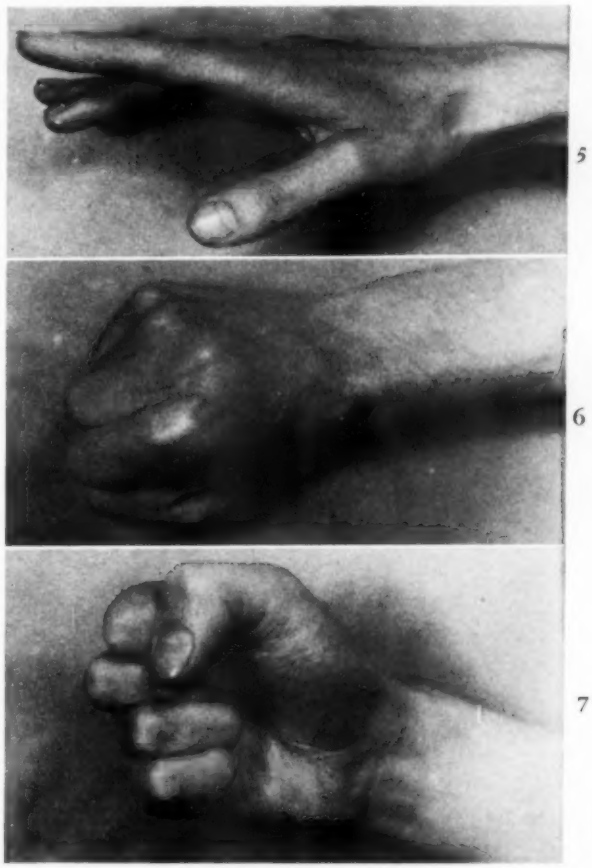

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Fig. 5. Reveals some slight limitation in extension of the finger which since the taking of the photograph, has returned to normal.

Fig. 6. Shows distinctly the shortening of the second metacarpal and the finger but an ability to flex it at the metacarpophalangeal joint.

Fig. 7. Shows ability to close the second finger on the palm of the hand and make a fist in the ordinary manner which he was unable to do prior to the operation.

On the third postoperative week, all dressings were removed, the wound was healed at this time and he was started on passive and active motion.

April 26, 1935, it was noted that he had full motion in flexion but a limitation of 10 per cent in extension.

August 8, 1935, he was discharged having a good useful finger.

The accompanying photographs show the after results of this operation.

MANAGEMENT OF LARGE SKIN FLAPS*

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THE recent visit of Harold Gillies has released us from the necessity of defending the use and transfer of large amounts of full thickness skin and massive fat flaps as hazardous and uncertain procedures. The argument for a tubed pedicle or a delayed flap is a matter of the individual case and often is best solved by a combination of the two. It is seldom that two burn cases are alike, except that the areas which might best be used for repair have usually been destroyed.

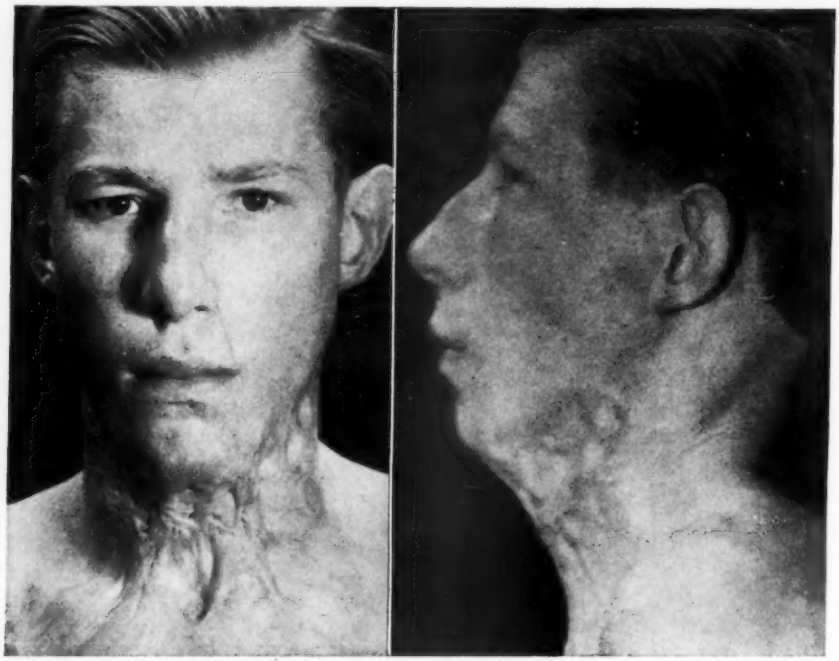

FIG. 1. FIG. 2.

FIG. 1. Old burn of neck; scar extends from chin to sternum with complete muscular inhibition.
FIG. 2. Complains of pain and difficulty in breathing on effort to raise chin.

Massive direct and jump flaps are indicated especially where difficulty in applying postoperative pressure eliminates full thickness grafts. They are also the choice where exceptionally large areas are to be replaced and where the underlying flap fat may be an aid to the final appearance rather than a danger to a tube. In estimating tissue replacement by massive direct and jump flaps, one must figure on a delayed flap to insure circulation. There must also be estimated the amount of retraction of the area to be replaced, once the offending or marginal tissue has been removed. To this must be added flap contraction, especially in the jump flaps. Allowance, too, must be made for tissue lost in trimming the margins for correct approximation.

Remembering these points, one measures the defect first plus the above allowance. A cloth or leather model is then made and

* Read before the Society of Plastic and Reconstructive Surgery, Atlantic City, June 14, 1935.

the flap procedure rehearsed in reverse to insure enough slack to prevent tension after suturing.

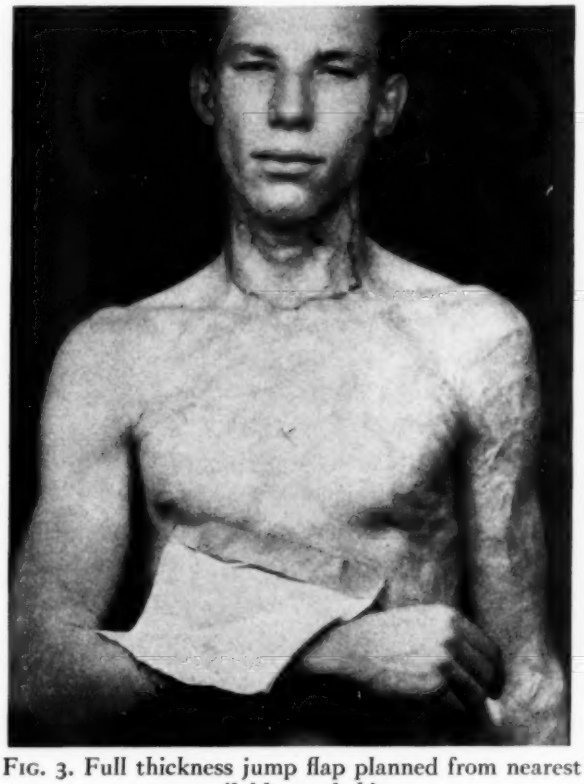

Fig. 3. Full thickness jump flap planned from nearest available good skin.

The less raw area exposed between stages, the less danger of secondary infection. The area from which the flap is planned should therefore be chosen with the thought of quick and easy coverage by thin grafts.

These generalities can best be reduced to their components by actual cases.

My first case (Fig. 1) is a boy of eighteen years who was badly burned in a gasoline explosion as a child. The neck, chest, back and upper arms were involved. The chin had been allowed to heal to the sternum (Fig. 2) and the neck muscles as a result were markedly atrophied. The preliminary work consisted of freeing the neck adhesions and inserting a large stent inlay covered with a thin graft. After the stent was removed he was discharged under the guidance of physical therapy for six months for rejuvenation of the neck muscles.

The nearest available unscarred skin, (Fig. 3) in an adequate amount for a flap was the upper abdominal region. Measurement of the neck

Fig. 4. Skin on forearm has been raised and used to line the raised lower half of flap.

scar area needing replacement was 8 by 10 inches. After figuring the excess tissue estimated for contraction of the flap, retraction of wound edges and excess margins, a flap 10 by 14 inches was outlined and incised on the upper abdomen. The lower half of the flap was elevated and resutured. Three weeks later a flap 4 by 12 inches was elevated from the right forearm (Fig. 4) and joined to the lower part of the abdominal flap. The abdominal defect was then thin grafted. Three weeks later the flap was lifted in its entirety and folded over on itself on the arm (Fig. 5). The remaining abdominal defect was grafted and the patient discharged for a month following a ten day hospital stay.

On reentrance the upper portion of the neck was dissected free and resutured to the middle third of the unrolled arm flap, the upper third of which was attached to the upper neck (Fig. 6). In three weeks time the rest of the neck was denuded and the flap fully unrolled, the arm skin returned, with the result which you see (Figs. 7 and 8).

FIG. 5. Flap has been folded over on itself. Full size regained by dissection when ready for use.

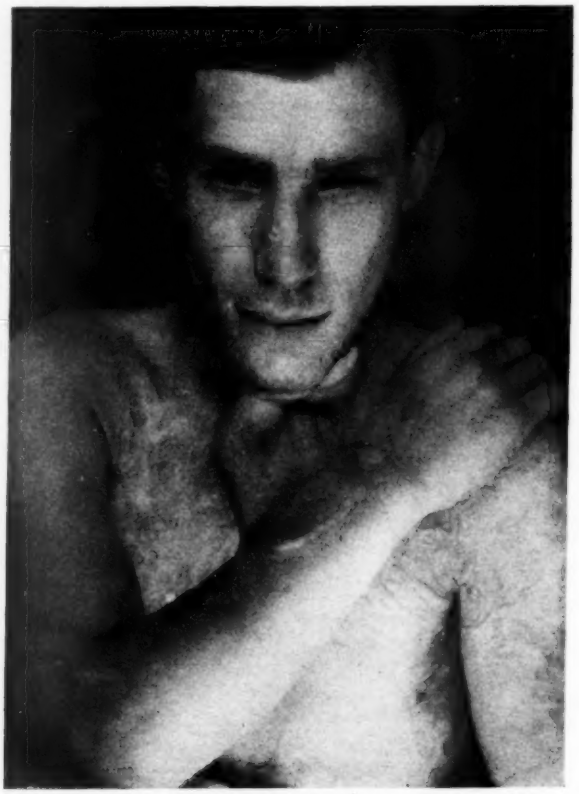

FIG. 6. Portion of flap has been attached to skin.

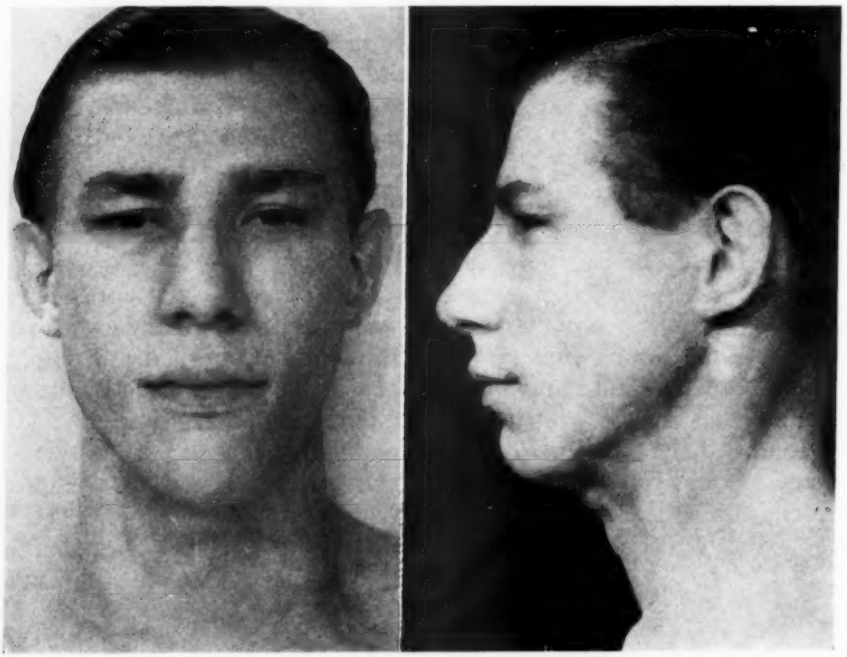

FIG. 7. FIG. 8.

FIG. 7. Final result. Abdominal defect repaired by thin grafts from leg.
FIG. 8. Restoration of function and cosmetic detail.

Gillies spoke recently of a large tube flap transferred from the abdomen to the leg, which finally left the navel on the knee. About a year ago I saw a patient who had had very large breasts and who had survived a five stage operation in an office for breast reconstruction. The physician had attempted to amputate the breast tissue, feeling that the breasts were too large and lumpy for a mastopexy, leaving very large and thick skin flaps to form new breasts. To insure a cosmetic result he constructed two tube flaps bearing the nipples at the ends, to be planted higher on the chest. Everything had sloughed according to nature. Some time later a clinic case presented itself with very large benign, hypertrophied and persistently lumpy breasts, with a history of several children and no milk. She had been advised amputation but naturally desired a reconstruction if possible. I remembered that Thorek had suggested a procedure in 1922 which offered a solution. Accordingly I grafted the nipples as free full thickness grafts using a dressing of vaseline-parresine gauze held in place by flat oval pieces of stent, over which stay sutures through the grafts and surrounding skin had been tied.

Three weeks later large skin flaps much thicker than usual and bearing the new nipples were elevated from over each breast. All of the breast tissue was amputated allowing a complete examination of all lumps. There was more than sufficient skin and fat for an acceptable reconstruction.

Summing up this brief paper, the most important factor in the management of large skin flaps, is the careful planning for each case, well ahead of the actual surgery.

IDIOPATHIC ENTEROSPASM OF ENTIRE ILEUM AND LARGE BOWEL*

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IDIOPATHIC enterospasm is an entity of rare occurrence. The diagnosis which is made at operation or autopsy is determined by evidence of spasm and the exclusion of other pathological findings. Since no apparent cause for the spastic bowel is evident, the condition has been classified as "idiopathic." The causes suggested have been entirely theoretical since autopsy has not revealed any information which might offer a possible etiology. There are no age limitations, cases having been reported from three and one-half months to seventy years in both sexes. About 160 cases have been recorded in the literature. Spastic ileus has been encountered in both the large and small bowel or only in a small segment of either, but the terminal ileum is the most frequent site of occurrence. The involved part is contracted to 0.5 cm. in diameter, the serosal luster is diminished, ischemia is present even to the degree of a bluish hue and the intestine is unyielding to the touch. The proximal and distal ends of the obstruction may be distended or normal, and in a few cases the proximal end has hypertrophied. However the transition between spastic and normal bowel is commonly abrupt. During operation spasm may persist, be intermittent or disappear with or without the application of heat. The symptomatology resembles that of an acute intestinal obstruction.

CASE REPORT

A. R., a white male, aged forty-two years, occupation bricklayer, was admitted by ambulance on September 25, 1932, complaining of acute abdominal pain. About seven hours before admission, following a bowel movement, the patient was suddenly seized with severe cramp-like pains in the hypogastrium which became more severe during the day. There was no vomiting.

The past history was entirely irrelevant except that the patient had lost 10 pounds during the last two months.

Physical examination revealed a well nourished male appearing in acute pain; temperature 99.6, pulse 80, respiration 26, blood pressure 130/84, leucocyte count 13,280, polymorphoneutrophiles 78 per cent, and small lymphocytes 22 per cent. The abdomen was entirely rigid with marked tenderness, hyperesthesia and rebound tenderness; the liver dullness was obliterated. No positive findings other than the local condition were ascertained. The provisional diagnosis was generalized peritonitis due to a ruptured peptic ulcer and laparotomy was performed under gas-oxygen-ether anesthesia a short time after admission. A right rectus incision was made. There was no free peritoneal fluid. The appendix appeared normal and was removed by the carbolic knife and ligature method. The entire large gut and the small intestine up to the jejunum was found collapsed in a state of spasm, ending abruptly at the jejunum, about 0.5 cm. in diameter and appeared blue in color. The mesentery appeared normal. The bowel recovered with the application of warm saline. The gall bladder was thin walled and emptied easily. The stomach and duodenum showed no pathology. With the index finger through the Foramen of Winslow, palpation revealed a moderately fibrous pancreas. The kidneys were normal in size and shape. A jejunostomy after the method of Witzel was performed 6 inches proximal to the ligament of Treitz employing a mushroom catheter. Abdominal closure was made in layers.

Signs of pulmonary complications were manifested on the first postoperative day and

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x-ray pictures revealed a tuberculosis involving the apices. The patient improved rapidly and seemed well on the road to recovery. The enterostomy tube functioned satisfactorily and distention was absent. On the third day, the patient suddenly expired, death being attributed to cardiac failure. Autopsy was not obtained.

COMMENT

The prominent features in this case were the absence of intestinal symptoms prior to admission to the hospital; the absence of abdominal pathology except for the spasticity of the intestines; the absence of postoperative distension. For lack of further evidence, death was attributed to cardiac failure. The picture at operation was typical of the entity described as an idiopathic spasticity of the intestines. Our case differed only in that it similated a ruptured peptic ulcer instead of the usual intestinal obstruction.

SUMMARY

A case of idiopathic enterospasm of the entire ileum and large bowel is presented. The operative findings, marked spasm of the gut and the absence of any definite pathology, were typical of this condition. Death was apparently due to heart failure and not to intestinal obstruction as is occasionally reported.

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SPONTANEOUS RUPTURE OF URINARY BLADDER

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CROSBIE,[1] in 1924, said that "spontaneous rupture is very rare and almost never occurs, except where there is disease of the walls of the bladder." In the only case which he had encountered the rupture was secondary to a huge carcinoma of the bladder. By spontaneous rupture is meant that type which occurs without external stimuli, the exciting cause, regardless of the nature of the rupture being unquestionably internal. Rupture of the bladder per se, secondary to penetrating wounds, was familiar to the ancients. Homer described the death of Phereclus as the result of a wound from a spear which passed from the buttock, through the perineum and into the bladder. Spontaneous rupture of the bladder, however, was not recognized until some centuries later, Pierus[2] reporting the first case in 1279. The next recorded case of spontaneous rupture was reported by Johnstone in 1773.[3] Since that time several cases have been added.[4–9] In 1931, Stone[10] found 40 cases reported in the literature, to which he added 2.

The factors which cause such a lesion are manifold and the classification presented by stone is very complete (Table 1).

Despite the many lesions which may produce this pathologic condition, it is seen only occasionally.[11] At the Cook County Hospital, between 1889 and 1893,[12] only 5 instances of all types of rupture of the bladder were seen among 8000 patients admitted to the surgical service; at the Rhode Island Hospital, from 1868 to 1929, 2 instances of spontaneous rupture were found among 226,632 patients admitted to the medical and surgical services, and at the Episcopal Hospital, of Philadelphia, from 1900 to 1905, 3 instances of intraperitoneal rupture were found among 8367 patients.

TABLE 1

STONE'S CLASSIFICATION OF SPONTANEOUS RUPTURE OF THE BLADDER

A. Inflammatory lesions of the wall of the bladder
   1. Intramural origin
   2. Extramural origin
B. Malignant disease of the wall of the bladder
   1. Intervesical
   2. Intravesical
   3. Extravesical
C. Obstructive changes at the neck of the bladder
   1. Prostatic enlargement
      (*a*) Benign
      (*b*) Malignant
      (*c*) Inflammatory (including seminal vesiculitis)
   2. Calculi
   3. Paralytic
   4. Uterine and adnexal disease in the female
   5. Occurring during labor
D. Obstructive lesions in the urethra
   1. Strictures
   2. Valves diverticula
   3. Tumors
   4. Calculi
   5. Peri-urethral inflammation
   6. Peri-urethral tumors

Rupture of the bladder, whether traumatic or spontaneous, is either intraperitoneal, extraperitoneal, or both, with obstructive changes at the neck of the bladder being the commonest findings. Intraperitoneal rupture occurs about twice as frequently as does extraperitoneal rupture, if the etiologic factor is obstruction. However, if the pathologic change is primary in the vesical wall, the extraperitoneal type predominates, in the ratio of about 2:1. The usual situation of intraperitoneal rupture is the vault, in the midline.[13] This is expected when one considers that this situation is probably the weakest point in the musculature, from an

embryonic standpoint, as well as being the farthest removed from the supporting pelvic tissues and framework. Intraperitoneal rupture usually extends directly through all the coats of the wall of the bladder and the tear in the peritoneum is adjacent to that in the bladder, but is not as large as the tear in the wall of the bladder. Frequently, the opening into the peritoneum is only a small fissure, and it was for this reason that A. T. Cabot,[14] in 1891, advised that exploratory laparotomy should be performed, so that the presence of urine might be detected. The opening in the wall of the bladder usually is ragged, and only rarely will microscopic examination reveal any pathologic changes in the tissue.

The symptoms which are produced depend primarily on whether the lesion is intra- or extraperitoneal. In either type, the history is of unlimited value. The extraperitoneal rupture produces pain and swelling above the pubis; the swelling soon becomes purulent. Small amounts of urine are passed, which, on gross or microscopic examination, are found to contain blood. Shock and its sequela appear rather late. According to Pedersen,[15] a bluish discoloration and tenderness about the umbilicus are symptoms of extraperitoneal rupture. Extraperitoneal rupture which occurs late may be converted into intraperitoneal rupture by perforation, secondary to changes caused by the urine or pus acting on the outer surface of the perforation.

The intraperitoneal rupture is far more grave than is extraperitoneal rupture, and early diagnosis is more urgent and difficult in the intraperitoneal than it is in the extraperitoneal type. Usually the first evidences of rupture are sharp abdominal pain and some shock, which may last only a short time; the patient feels better after the symptoms disappear, and, as a result, does not consult a physician for some hours. Urgency of micturition follows in most instances; only a few drops or no urine are voided. At first, the abdomen is very rigid or ligneous; later, the tenderness becomes localized, particularly in the upper portion of the abdomen. The presence of fluid is evidenced by a shifting dulness in the flank.

The most important element in establishing the diagnosis is the history, carefully taken, which coupled with a thorough physical examination usually serves to establish a diagnosis. The advisability of checking a known quantity of solution injected into the bladder with the amount returned, is no longer considered the diagnostic method of choice. Martin[16] advised the use of a cystogram for detecting rupture of the bladder, while Herbst[17] advocated cystoscopy, maintaining that evidence of the rupture and its location will be obtained by the injection of fluid, as the opening into the peritoneal cavity occasionally is sufficiently large to permit all of the fluid which has been introduced to return through the catheter.

Once the diagnosis has been established, a surgical procedure must be done for either the intraperitoneal or extraperitoneal rupture.[18,19] Cystostomy is all that is required in a case of early extraperitoneal rupture. In cases in which the rupture has been present for some time, and in which urinary extravasation has occurred, multiple incisions are necessary. In the intraperitoneal type, a thorough inspection should be made of the abdominal cavity, and it, as well as the bladder, should be drained. It seems to be the opinion that the tear in the bladder need not be sewed, as long as adequate drainage is afforded to the bladder. This was first mentioned by A. T. Cabot, in 1891, and later, by Thomas[20] and by Crosbie.[1] If doubt exists as to whether the rupture is intraperitoneal or extraperitoneal, an exploratory laparotomy is always advisable.[21] Nicolaysen[22] has reported a case in which cure was obtained by means of drainage with the catheter alone.

REPORT OF CASE

A man of Jewish descent, aged forty-seven years, came to the clinic August 26, 1935, because of a postnasal dripping and fatigue

which had followed a nervous breakdown in the spring of 1935. In the course of his illness he had been very irritable, despondent, and forgetful. Physical examination at that time had not revealed any abnormality.

Physical examination at the clinic did not reveal any abnormality except a small, indirect inguinal hernia on the left side, which had been present for three or four years. Loss of weight had not occurred; the patient weighed 140 pounds (63.5 kg.). The value for the blood pressure, expressed in millimeters of mercury, was 100 systolic and 70 diastolic. Examination of the ocular fundi did not reveal any abnormality.

On neurologic examination, the left pupil reacted sluggishly to light, and the speech was slurred somewhat to alliteration. The deep reflexes, particularly the knee jerks, were hyperactive, grade 2. The patient was argumentative, a poor calculator and had a poor insight.

The specific gravity of the urine was 1.030; the urine contained albumin, grade 2+; erythrocytes, grade 1+, and a few leukocytes. The value for the hemoglobin was 15.4 gm. per 100 c.c. of blood, the erythrocytes numbered 4,960,000 and the leukocytes, 10,500. A differential count revealed 20 per cent lymphocytes, 12 per cent monocytes, 67 per cent neutrophils and 1 per cent eosinophils. Examination of the serum disclosed that the Kline test was positive, grade 1+; the Kahn test was positive, and the Hinton and Kolmer tests were negative. On examination of the spinal fluid, the Kline test was positive, grade 4+; the Nonne-Apelt reaction was positive, and the Wassermann reaction was strongly positive. There were 51 cells per cmm. of spinal fluid, and the colloidal benzoin curve was 133 333 333 322 000. The guaiac and benzidine tests revealed the presence of occult blood in the feces.

Roentgenologic examination of the thorax disclosed that the pleura was slightly thickened at the left apex.

A diagnosis of syphilis of the central nervous system was made and malarial treatment was instituted August 18, 1935. The temperature rose to 105.4°F. (40.7°C.) on that day and again on August 20 and 21. Administration of quinine was commenced August 21 and was continued for forty-eight hours. The patient felt well and was up and about in the hospital from August 22 to August 26.

At 5:00 A.M. on August 26, 1935, the patient was awakened from a sound sleep by severe pain in the upper portion of the abdomen. He was slightly nauseated but did not vomit. The temperature was 97.8°F. (36.5°C.), the pulse rate was 94 beats per minute, and the value for the blood pressure, expressed in millimeters of mercury, was 100 systolic and 70 diastolic. There was increased resistance over the entire abdomen, but it was more marked over the upper portion with evidence of fluid obtained by a shifting dulness to percussion. The nature of his condition was problematic, and it was thought best to study his progress for the following two hours. A catheterized specimen of urine revealed the specific gravity to be 1.010; albumin, grade 2+, erythrocytes, grade 2+, and 8 leukocytes per high power field. The erythyroctes numbered 3,880,000, and the leukocytes numbered 12,800 and the value for the hemoglobin was 14.9 gm. per 100 c.c. of blood. During the four hours immediately after the onset of symptoms, shock had occurred. The abdomen became much more rigid and boardlike than it had been. It was thought that perforation of a viscus had occurred, and exploratory operation was performed.

Spinal anesthesia was given and the abdomen was opened through an upper right rectus incision. When the peritoneum was opened, a large amount of straw-colored fluid was found in the abdominal cavity. A tear, about 2 inches (5 cm.) in circumference, was found on the posterior-superior surface of the bladder. The peritoneal opening was about the same size as, and adjacent to, that in the bladder. The opening in the bladder was closed with sutures of No. 2 chromic catgut. The bladder was drained extraperitoneally by means of a split tube which was inserted into the space of Retzius. A Penrose drain was placed in the pelvis to drain the abdomen, and a Pezzer catheter was inserted into the bladder.

Convalescence was uneventful until the eleventh day, at which time the upper angle of the incision spread sufficiently to allow a knuckle of bowel to escape between the silkworm gut sutures. A secondary closure was made by using through and through silkworm sutures. The subsequent course was entirely uneventful and the patient was dismissed from the hospital on the twenty-eighth day following the second operation. At that time, he was able to empty the bladder completely. During

convalescence, 0.4 gm. of neoarsphenamine was administered once a week.

In a letter received three weeks later, the patient stated that he apparently was in splendid health.

SUMMARY

A case of a ruptured urinary bladder is reported because of the rarity of the pathologic condition. It also emphasizes the importance of surgical interference in acute abdominal disorders when certain signs, such as acute pain and generalized rigidity are present. The character of the fluid in the abdominal cavity, although it is no different in appearance than that frequently found in cases in which ascites occurs as a result of a variety of causes, suggested the possibility of urine from a ruptured bladder.

The cause of rupture of the bladder in this case is not known. The patient said that he had voided 3 or 4 ounces (90 to 120 c.c.) of urine soon after the onset of the acute pain in the upper part of the abdomen, and that he had voided before retiring, eight or nine hours previously. The urine obtained by passage of a catheter three or four hours before operation evidently came from the abdominal cavity. The edges of the opening in the bladder were ragged and appeared necrotic. They were trimmed until healthy appearing tissue was reached. Unfortunately, the excised portion of the vesical wall was not preserved for microscopic study. Syphilitic ulcerations do have a tendency to occur in the midline, for instance, ulcerations in the midline of the anterior thoracic wall and sternum are seen occasionally. In such instances, syphilis is always to be ruled out as a possible etiologic factor. In this case, the rupture was in the midline, and obstruction of the neck of the bladder was not a factor, as it previously has been reported to be in some of the cases.[23]

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FRACTURE OF ANTERIOR SUPERIOR SPINE OF ILIUM

A SUMMARY OF THE LITERATURE AND REPORT OF A CASE*

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FRACTURE of the anterior superior spine of the ilium due to muscular violence is rare. In recent years however the condition is being more frequently recognized. Up to 1924 Carp[1] collected 21 cases, including a case of his own, of fractures of the anterior superior spine of the ilium. Christopher,[2] in 1933 was able to find 23 additional cases and added one of his own, making a total of 45 cases. Since then we have been able to collect 12 other cases in the literature.[3–8] Seven of these were published immediately prior to Christopher's report.[3,4] Our case, accordingly, makes 58 to be reported up to this time. In reviewing the literature we have included only the cases which were caused by muscular violence, however, only one case was found which was not caused by such force.

According to Carp there has been some question as to whether this is an epiphyseal separation or a fracture. He states that McHenry and Turner believe the anterior superior spine has only a center of ossification, which is secondary to a center, from which the whole iliac crest arises. Epiphyseal separation therefore, which does not include the whole iliac crest, would not be considered a true epiphyseal separation. Carp concurs in this opinion. Fracture therefore would seem to be the more correct term.

Carp quotes Poland as stating that the center of ossification for the anterior superior spine, secondary to that of the crest of the ilium, appears at about the fifteenth year. It does not become united to the ilium until the twentieth to the twenty-fifth year, usually the twenty-first year. According to this, as Carp points out, a fracture or separation of the true osseous epiphysis can occur only from the fifteenth year, the time of its formation, to the twenty-fifth year, the time of its union with the body of the bone. It is a fact that practically all cases have occurred between these ages.

The majority of all cases have occurred in boys while participating in some type of athletics. For this reason it has been termed a typical sport injury or as the Germans term it "Sportverletzung." A few cases, however, have occurred in girls and Carp quoted one case as occurring in a man seventy years of age.

The most important muscles having attachment to the anterior superior spine of the ilium and having to do with this fracture, are the muscle tensor fascia lata and the sartorius (Figures 1 A and B). The action of the former is to tense the fascia lata and assist in flexion and inward rotation of the thigh.[9] The action of the latter is to assist in the flexion of the thigh, flex the lower leg and rotate it upward when flexed.[9] Poupart's ligament is also attached to the anterior superior iliac spine and probably exerts an influence on it because of the muscles attached to it; the internal and external oblique, the transversalis and fascia lata.[10]

The mode of production of this injury is of particular interest. Most authors agree that the pull of the sartorius and tensor fascia lata causes the fracture, but a few think that the tensor fasia lata exerts the stronger force in this connection. The majority however think the sartorius exerts the greater force.

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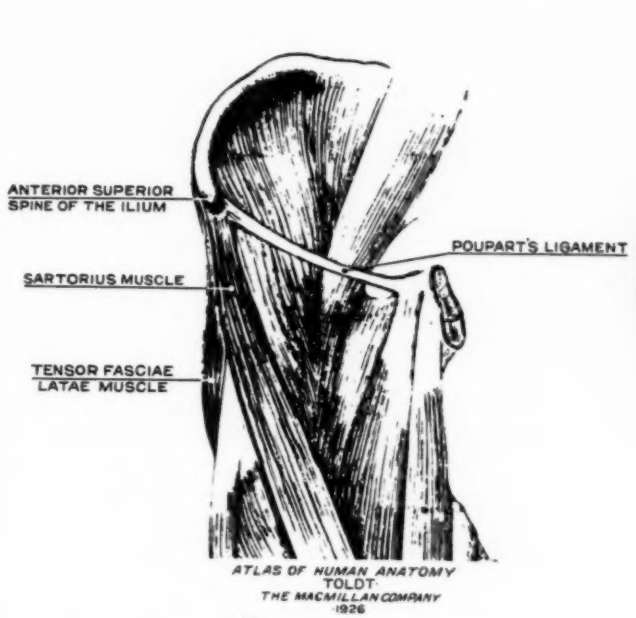

FIG. 1A.

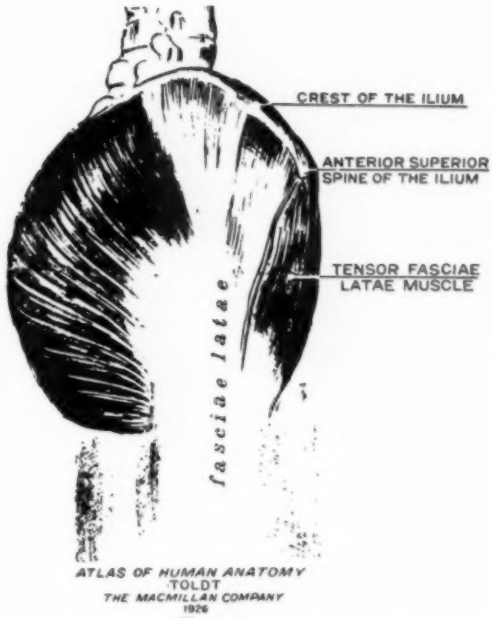

FIG. 1B.

FIG. 1. A. Sketch showing the muscles attached to the anterior superior iliac spine, which by their forceful contraction cause fracture of this structure. B. Sketch showing the anatomy of the tensor fascia lata muscle in relation to the crest of the ilium and anterior superior iliac spine. (From Toldt's Atlas of Human Anatomy.)

FIG. 2A.

FIG. 2B.

FIG. 2. A. Radiograph, taken eighteen hours after injury showing fracture of the anterior superior spine of the ilium. B. Radiograph ten months later, after conservative treatment, showing solid bony union of the fracture.

The fracture usually occurs, of course during extreme physical effort, and usually in the act of some sudden movement. The injury occurs when the trunk is hyperextended on the thigh or vice versa. Any sudden movement as in trying to straighten up quickly, when the trunk is hyperextended, as occurs when one slips and is about to fall, or when wrestling, may bring the sartorius and tensor fascia lata into sudden and powerful contraction and result in the avulsion of the anterior superior spine of the ilium. On the other hand, when the thigh is hyperextended on the trunk, as in running, or kicking a football and the thigh is suddenly flexed the same sudden forceful muscular action may take place. Indeed, most cases have resulted from some of the following actions: running, jumping, wrestling, kicking a football, slipping, sudden turn backward and sudden incoordinated movement.

We believe that due to the greater strength and more powerful action of the sartorius in flexing the thigh, and due to the fact that the fragment is nearly always displaced considerably downward in the direction of the pull of the sartorius, this muscle unquestionably furnishes the predominating force which causes the fracture.

The diagnosis depends upon the history of a sudden pain in the region, the onset occurring during physical effort, local pain, tenderness, swelling and crepitation and a confirmation by the roentgenogram.

Various forms of treatment have been tried. The results, according to case reports, have been uniformly good, whether treated conservatively, ultra conservatively or radically. Some cases have had only rest in bed, others have been strapped in various ways with adhesive, still others have been immobilized in plaster, while many have been reduced by open operation. Since conservative methods usually give as good results as an open reduction, even though accurate anatomical reduction is not often as good in the former method as in the latter we think that the closed treatment is the method of choice in the majority of cases. The procedure used in the closed method of treatment are immobilization for one month in a plaster spica with the thigh and leg flexed with no resumption of any strenuous exercise until after six months or more.

REPORT OF CASE

C. B., white male, aged seventeen years was admitted to the Orthopedic Service of the Employees Hospital September 28, 1934, about eighteen hours after receiving an injury while playing football. He was running at full speed with the ball when suddenly he tried to side step a would-be tackler. He was seized immediately with a sudden excruciatingly acute pain in the region of the left anterior superior spine of the ilium, he was tackled instantly after the pain began and had to be carried off the field.

When first examined upon admission to the hospital he was lying on his right side in bed with his legs and thighs flexed, which was the position of most comfort. Any attempt to move caused considerable pain but by careful manipulation the left thigh could be extended. There was acute pain, tenderness and moderate swelling over the left anterior superior spine of the ilium. A loose fragment could not be palpated because of the swelling and tenderness. Radiographic examination (Fig. 2A) revealed a fracture of the anterior superior spine of the left ilium. The detached fragment was displaced downward about 1.5 cm. and laterally about 0.5 cm.

On account of the marked swelling and tenderness over the involved area an overhead fracture frame was applied to the bed and the left lower extremity was suspended with the knee and hip flexed at 45° by means of a towel which was passed under the knee and suspended by a rope and weights. Local heat was also applied. Four days later a plaster spica was applied to the left hip with the thigh and knee flexed at an angle of 45°. The cast was removed about six weeks later when guarded active and passive motions were begun with hot baths. He was discharged from the hospital four days later with no pain. He began walking with the aid of crutches, was seen again in two weeks when function was good and there was no limitation of motion in any direction or pain on motion. The patient was last examined on August 5, 1935, ten months after the original injury. At this time all move-

ments were normal and there was no pain on exertion. Another radiograph (Fig. 2B) revealed a complete healing of the fracture by solid bony union. He could do anything in the way of athletics that he did before the injury. He participated in football, basketball and track during the past year, and his exploits on the gridiron were legion, gaining him wide acclaim.

SUMMARY

A review of the literature reveals that fracture of the anterior superior spine of the ilium is an uncommon injury. It occurs in the majority of instances in the young athletic individual from seventeen to twenty-five years of age. The direct causative factor is the pull exerted by the sartorius and tensor fascia lata.

The diagnosis depends upon the history of sudden, severe pain in the region of the anterior superior spine of the ilium, occurring during an act of physical exertion. The usual localized signs and symptoms of a fracture are present. Roentgen examination confirms the diagnosis.

A case is reported which is the fifty-eighth to be found in the literature. Excellent results were obtained by conservative treatment.

The best treatment is immobilization for four weeks in a plaster hip spica with the thigh flexed on the trunk. Active physical exercises should not be resumed for six months or more.

We do not believe that open reduction is indicated in the average case. Even though accurate anatomical reduction is not always accomplished in the closed method, the end results are just as good.

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PERICARDIOSTOMY FOR ACUTE PURULENT PERICARDITIS

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THE patient is a six year old female who ten days before admission to the hospital developed a head infection and a cough. After several days, there was an onset of chills and fever and the patient complained of pain in the right side of the chest; on the day before admission the attending physician found signs of fluid. Respirations rapidly became increasingly embarrassed so that at the time of admission to the hospital there was a very marked dyspnea.

The family and previous history of the patient were both irrelevant and of no importance as far as the present illness was concerned.

Physical examination showed a very dyspneic child, markedly toxic, with a temperature range between 104°, and 105°F. Except in the chest there were no elicitable objective findings. In the thorax there were absent breath sounds, fremitus and voice over the entire right chest and the left border of the heart was at the left anterior axillary line.

The hemoglobin was 75 per cent. A blood culture at this time showed eighteen colonies of the gamma type of the non-hemolytic streptococcus. The same bacteria were found in the urine and in the stools. Evidently the child had a relatively severe general infection of which the pleural effusion was a manifestation.

An *x*-ray examination confirmed the presence of fluid in the right chest and the marked displacement of the heart and mediastinum to the left.

In view of the fact that the accumulation of fluid in the chest was of such short duration that the heart was displaced so far to the left and because of the tremendous toxicity, preliminary treatment of daily aspirations of the chest were done and for three consecutive days between 500 and 600 cc. of frankly purulent fluid were withdrawn. The fluid contained large numbers of the alpha type of Streptococcus Viridans. By the fourth day the heart shadow had retracted to its normal position, the toxicity had lessened markedly, and the dyspnea had lost much of its labored character and believing that the patient had been adequately prepared operation was undertaken.

Under local anesthesia an intercostal incision was made in the posterior axillary line between the eighth and ninth ribs on the right side. Closed drainage was established under water.

The child continued to do well, but the temperature and other signs of general infection still continued. On the twelfth postoperative day a small abscess of the left metatarsal region was incised and drained. Radiographic study of the foot showed no bone involvement. On the fifteenth postoperative day the blood culture became sterile and remained so thereafter. The clinical picture however did not change as yet and the signs of general infection persisted.

At this time the *x*-ray study of the chest showed that the empyema cavity occupied two-thirds of the chest space on the right side; following the operation it was adequately drained and that the heart and mediastinal shadow were in normal position. Nevertheless, the dyspnea showed signs about this time of becoming progressively aggravated. On the eighteenth postoperative day the *x*-ray demonstrated an enlargement of the pericardial shadow and confirmed a suspicion of a pericardial effusion which the physical signs had indicated. The effusion was proved by an aspiration of the pericardial sac of a golden yellow, slightly turbid serous fluid with some fibrinous flakes and contained Streptococcus Non-hemolyticus.

The general condition of the patient at this time was still good. There was considerable dyspnea indicating a condition of heart tamponade, but there was very little, if any, cyanosis or other sign of an embarrassed circulation. The available facts included: (1) an adequately drained empyema cavity; (2) signs of continuing general infection and of a local metastatic focus in the pericardial sac; (3) signs of heart tamponade which seemed to be increasing; (4) the sum total indicating a serious state of affairs the urgency upon which Dr. Lilienthal insisted. The relative

merits of aspiration of the pericardial effusion and open operation were freely and adequately discussed; and operation favored in spite of the fact that the pericardial exudate was a relatively thin, turbid, serous fluid.

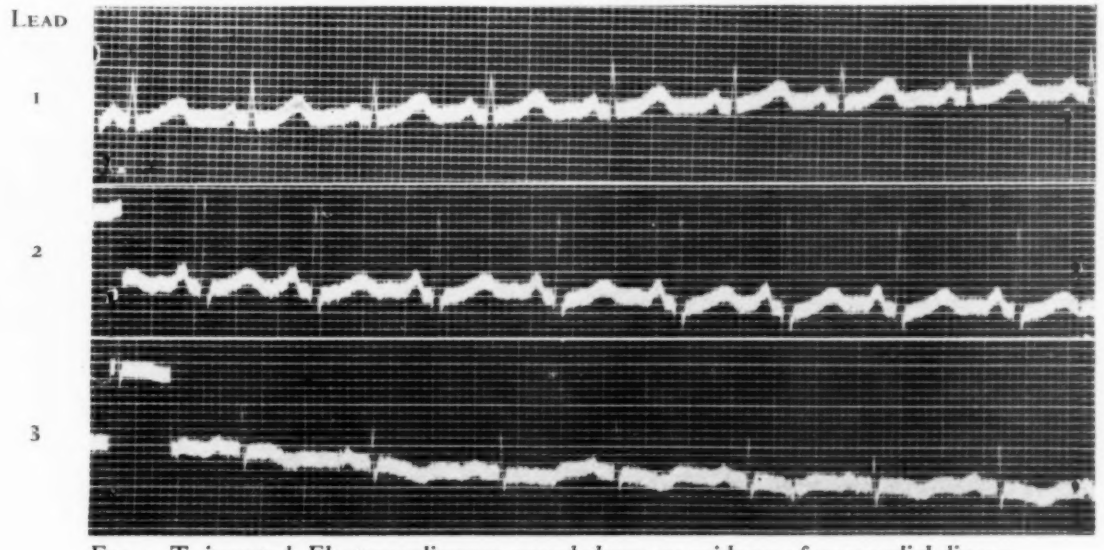

Fig. 1. T_3 inverted. Electrocardiogram record shows no evidence of myocardial disease.

The child was exceedingly cooperative and the operation was done under local anesthesia; she submitted quietly and with astonishing fortitude.

The question of draining the pericardial sac from either the right or left side of the sternum was also discussed and the right side was chosen because the empyema involved this side and operation on the left side carried with it the danger of entering into and infecting the intact left pleura.

A vertical incision was made at the right border of the sternum, adequately exposing the areas and then resected the fifth and sixth right costal cartilages. The tissue carrying with it the anterior edge of the right pleura was bluntly pushed away until the pericardium was seen. A little more room was now gained by biting away with the rongeur forceps some of the sternal tissue. The pericardial sac was then aspirated and when fluid was obtained an incision about 1½ inches in length following the course of the needle was made with a narrow scalpel. About a pint or more of turbid serous fluid escaped through this opening. The edges of the pericardial incision were fastened to the skin with several catgut sutures and no drain of any kind was inserted.

The little patient reacted well to her operation with very little shock. In the postoperative care a point was made of taking the child in ones arms at frequent intervals and turning her face downwards to encourage the escape of pericardial exudate. A considerable amount escaped in that way especially during the first few days after the operation.

There was progressive improvement thereafter, the dyspnea lessened gradually; first noticable when the patient lay quietly in bed but later when she was disturbed, moved or even turned upon her face. The wound was clean and showed signs of progressive healing.

The hospital cardiologist saw the patient on the tenth day after the pericardiotomy and the twenty-eighth day after the thoracotomy. His report is as follows:

"Pallor, moderate. Slight engorgement of the veins in the neck. No edema, no petechiae, no cyanosis or orthopnea. No ascites. Broadbent's sign negative. No thrills felt. Spleen not palpable and not enlarged on percussion. Liver normal. Left cardiac border at the axillary line (anterior). Heart sounds good, distinct. No murmur at the apex. P_2 greater than A_2. Rhythm regular, rate 140 per min. Right lung: feeble, breathing in lower parts; broncho-vesicular breathing over upper parts (compression). Left lung normal. Respiration 40 per min. Pulse responding to heart action; soft (toxic); regular. B.P. 90/55 E. Extremities warm.

"The absence of apical murmurs, of spleen enlargement, of petechiae, the clearance of positive blood cultures, lack of marked hema-

turia, and the general condition, indicate that we do not deal with any subacute bacterial endocarditis or with any carditis; that we are dealing with a suppurative pericarditis only, which fortunately for the child was diagnosed and operated upon in time. The electrocardiograph shows only some toxic effects (rapid rate) but no definite evidence of myocardial disease.

"The relatively good and free inflow of blood indicated by only a slight engorgement of the neck veins, the clear second heart sounds, the electrocardiographic findings, the blood findings, etc., indicate that the child has a good chance of recovery. In the left lateral position, the heart sounds are also distinct and clear indicating good drainage. The absence of carditis makes the prognosis more favorable, although it is still serious."

On the twenty-fourth, twenty-fifth and twenty-sixth days after the thoracotomy, and the sixth, seventh and eighth day after the pericardiotomy, there was an increase in the temperature. The *x*-ray study on the last day mentioned showed a moderate reaccumulation of fluid in the right chest. Further observation and *x*-ray studies, however, made us change that opinion and the visible shadow in the right chest was interpreted thereafter as a pleural thickening, very likely the result of some reinfection, possibly even from the pericardiotomy operation or wound. This at no time, however, played any role in the further clinical history of the case, except, possibly, as it was related to the next development.

On the thirty-ninth day after the thoracotomy and the twenty-first day after the pericardiotomy the temperature reached levels which varied between 99° and 101°F. and edema of the lower and posterior parts of the patient's body were noticed. Everyone agreed that this was due to some compression of the vena cava produced by an inflammatory exudation about its point of entry into the pericardial sac. Dr. Lilienthal suggested immediate exploration to relieve this compression. In two other similar experiences a reestablishment of the circulation occurred spontaneously the edema disappearing eventually. Besides, such exploration might readily prove fatal to the child in her present condition.

Three days later there was an improvement in the condition of the patient. Dr. Lilienthal concurred in this observation and agreed to the postponement of surgical intervention, but he maintained that eventually surgery might prove necessary, though I disagreed believing there would be complete spontaneous disappearance of the edema.

Up to this time and continuing until the thirty-third day after the pericardiotomy and the fifty-first day after the thoracotomy, the visible *x*-ray shadow of the heart did not change. On the fifty-fourth day after the thoracotomy and the thirty-sixth day after the pericardiotomy, the *x*-ray studies showed a marked decrease in the size of the heart shadow. Its pulsations were distinctly visible on fluoroscopy and the roentgenologist, Dr. Snow, interpreted this as indicating that the pericardial exudate was probably gone. This coincided clinically with the continuing improvement of the patient. The superior vena cava appeared dilated during the fluoroscopic observation but with each inspiration it decreased in width. Dr. Snow interpreted this as being caused by pericardial adhesions.

The child was now out of bed in a wheel chair. Improvement continued; the wounds healed by primary union and on the fifty-eighth day after the thoracotomy and the fortieth day after the pericardiotomy the patient was discharged from the hospital.

A follow-up observation on the ninty-sixth day after the thoracotomy and the seventy-eighth day after the pericardiotomy, showed the general condition of the patient was excellent, the color was good, there was no dyspnea, no clubbing of the fingers and no engorgement of the neck veins. The wounds were both healed solidly. There was no edema anywhere. The physical examination revealed no abnormalities either in the lungs and pleura or in the heart or pericardium.

The report of the follow-up roentgenographic study, made by Dr. R. A. Bendove is as follows:

"Fluoroscopic and *x*-ray examinations reveal both pulmonic fields well aerated. Slight peribronchial thickening throughout. Calcified nodules of various sizes in the right hilum region. Thickened right interlobar septum apparently between middle and lower lobes. Right costophrenic sinus is obliterated and the entire hemidiaphragm is somewhat flattened. However, it shows definite though limited respiratory movements taken in inspiration and expiration respectively. The

respiratory excursions of the left hemidiaphragm are of wide amplitude.

"Heart silhouette appears a little enlarged generally. Ratio between MR and ML is diminished. No evidence of enlargement of one particular chamber. The retro-cardiac space, as visualized in various oblique and lateral positions, is not encroached upon.

"Right phrenocardiac angle is blunt and partially obliterated. However, the alteration in size and shape of cardiac shadow in erect and supine positions point against any marked pericardiac-diaphragmatic adhesions.

"Upper mediastium is somewhat enlarged to the right, due probably to brachio-cephalic vessel-group and superior vena cava. It enlarges and decreases with respiratory phases, appearing wider during expiration in erect position.

"A few dense irregular patches in the cardiac area, in the left and right oblique positions. They suggest localized pericardial adhesions which do not interfere with cardiac movements as evidenced by the normal changes of the heart shadow in various positions and respiratory phases. Fluoroscopic examination shows rhythmic, forceful contractions, more pronounced on left.

"The electrocardiograph reveals no evidence of clinical pathology, except for tachycardia."

The follow-up report of the cardiologist, Dr. Kapp, is as follows: "The appearance was healthy, color good. There was no cyanosis, no engorgement of the veins on the neck, no edema, except a slight, hardly noticeable, puffiness of the left side of the face in certain positions; the right thigh was slightly thicker than the left (about 1 cm. difference) below the trochanters. There were no thrills, no abnormal pulsations at the apex or back. Broadbent's sign was negative. There was hardly any increase in the veins in the neck on Valsalva's experiment (deep expiration by closed glottis after deep inspiration). No dyspnea on moderate exertion.

"The left cardiac border was 2–3 cm. from the midclavicular which is rather due to some displacement of the heart to the left (because of right empyema with pneumothorax) and not to enlargement of the heart.

"There were no murmurs heard. The heart rhythm was regular; the heart sounds of good quality, clear and distinct. The electrocardium is negative except for some tachycardia. P2 was slightly louder than A2. The lungs showed vesicular breathing, and practically over the whole surface of the right lung. The liver and spleen were not palpable. There was no ascites. The blood pressure was: systolic 95, diastolic 65.

"Fluoroscopic examination: showed no dilatation of the large vessels, slight displacement of the heart to the left, no cardiac enlargement and good amplitude of ventricular pulsation. There were some pleuro-pericardial adhesions on the right side. The expansion of the right lung was very satisfactory, and the clearness at the costo-diaphragmatic angles was good.

"Diagnosis and Discussion: These findings clearly show that the obstruction to the venous return, found on previous examinations (with marked engorgement of the neck veins, swelling of liver, ascites, edema of lower extremities and face), has practically disappeared. The slight puffiness of the left side of the face might be due to the anatomical shape and position of the left jugular and innominate veins and to acuteness of angle of entrance into the vena cava superior. The slight swelling of the right thigh vessels, or possibly to some thrombotic phenomenon in the smaller vessels of the limb, although there was no history of pain in the leg and no tenderness on examination was noted at any time.

"The absence of any murmurs or any friction rub, and the negative electrocardiographic findings would justify the exclusion of any involvement of the endocardium or myocardium or of any active pericarditis are present. I therefore consider the results of the operative treatment as highly gratifying from the cardiological standpoint. Gradual exercise was advised. No medication necessary."

The second follow-up examination was made in June, 1935. The patient's mother stated that in March, 1935 she had consulted Dr. Joseph Eidelsberg because of increasing obesity. Examination of the child showed hypotonia, pot belly, coarse skin, etc., and the basal metabolic rate was minus 24 per cent. The impression was that the patient had a hypothyroidism due to exhaustion caused by the demands made upon her entire organism by the preceeding protracted illness. The patient received two grains a day of thyroid gland feeding; gradually increasing the dose to three and four grains. She tolerated the medication very well and under its influ-

ence there was a slight loss of weight and the basal metabolic rate rose to minus 13 per cent. Treatment was interrupted by the occurrence of an attack of measles.

In May, 1935 the basal metabolic rate had dropped to minus 22 per cent. The thyroid medication was resumed receiving four grains daily and the basal metabolic rate on June 17, 1935 was plus 6 per cent. On that date the weight was 73½ pounds and her condition in general was very much improved as to her skin, obesity, pot belly, hypotonia, etc. The pulse rate was 86 per minute.

It may be that the thyroid function will improve as the patient recovers from the effects of the infection and the exhaustion. It is probable that the thyroid gland administration will be gradually or diminished entirely.

DISCUSSION

There are numerous reports including a large number of cases, giving the necessary statistical information, best review being that of Winslow and Shipley who studied a series of 118 cases.

Pericardial purulent effusions, except for those few which follow gunshot and stab wounds are to be encountered as secondary or metastatic foci during some general infection, or as an infection by contiguity of structure when the pleura, lung or other neighboring structure is involved. Sometimes the latter two mechanisms are so intimately involved as to make it difficult to make the correct interpretation. The important point, however, seems to be that pericarditis and pericardial purulent effusions belong in the class of lesions which one must consider when other condition are present during the clinical course in which pericarditis is found; and, under such conditions, the presence of pericardial effusions must be watched daily. It is not to our credit that pericarditis and pericardial effusions, under the conditions mentioned, form one of the most frequent unexpected encounters in postmortem examinations.

In the case reported the pericarditis was a secondary manifestation during the course of a streptococcus general infection.

The commonest sequence follows some infection of the thoracic contents; the least common, perhaps, occurs after osteomyelitis of the sternum, not as a manifestation of the general infection, but as a local complication incident to the rupture of a subperiosteal abscess on the posterior aspect of the sternum into the pericardial space common after general infection.

The recognition of pyopericardium depends upon the medical preparedness of the attending physician, often repeated physical examination, and frequent roentgenographic study, which should always be confirmed by an immediate paracentesis. Of these three factors, paracentesis carries with it the danger of damage to the heart muscle or its nourishing vessels, and the danger of infecting new areas or hollow spaces, notably in the pleura.

The measure of the importance and the urgency created by an accumulation of fluid exudate in the pericardial sac is furnished by the degree and intensity of any accompanying heart tamponade which is present; the greater the heart tamponade the more urgent the necessity for its immediate relief.

The usual opinion is that the earlier the drainage is established the better for the patient and the more certain of the ultimate success of the operation. This opinion, however, does not seem to be substantiated out by collected statistical reports. For instance, in Winslow and Shipley's series between 55 and 61 per cent of the patients recovered regardless when the operation was done. And furthermore, in those operated before two weeks, 55 per cent recovered, while in those operated after two weeks 61 per cent recovered, indicating a better outcome in those not operated during the first two weeks.

Of the two available therapeutic procedures, paracentesis is only a palliative procedure and, if this is persevered in, alone, the mortality is 100 per cent. Some form of open drainage of the peri-

cardium is necessary. It is important to remember that the pericardium may be opened and drained from the right or left sides of the sternum or from below. The choice of right or left approaches enables one to avoid an infected side, as was done in the case reported and it seems that with this versatility of approach, the one from below is possibly superfluous.

Different forms of drainage apparatus have been employed, tubes and gauze forming the majority of the mechanisms employed. There are some reports in the literature of the pericardium having been sewn to the skin to form a wide cloaca, as was done in the recorded case. The result in this case was very satisfactory and would lead us to use this method again in any subsequent case.

Chemical sterilization of the pericardial cavity was not employed in this case. The experiences reported in the literature lead to the opinion that this method is of little importance in pyopericardium.

Secondary revisions of the wound are necessary sometimes because the formation of intrapericardial adhesions tend to form closed-off pockets, especially in the posterior and lower parts of the pericardial sac which do drain inadequately or not at all. Possibly, this tendency would emphasize one advantage of an early operation, in that the latter should be done before adhesions have had time to form. Nevertheless, the danger of a subsequent adhesive pericarditis seems to be only about 1.5 per cent. In this case, no secondary revision of the wound proved necessary and up to the present time there is no evidence of any adherent pericardium.

The prognosis seems to depend almost entirely upon the etiological factor involved, inasmuch as regardless of the time which elapses between the onset of the condition, or when the diagnosis is first made and the time of operation, the proportion of cures seems to maintain a fairly uniform level. This seems to hold true also for the method of establishing drainage. Primary infections of the pericardium seem to be followed by the best and most numerous recoveries. Pneumococcus infections are followed by about 50 per cent of recoveries. Streptococcus and staphylococcus infections are followed by the poorest results. There has been a gradual decline in the published mortalities from 60 per cent in the first 25 cases published to about 43 per cent for the last 28 cases.

SUMMARY

In a six year old female child a "catarrhal" infection was followed by an empyema in the right pleural space. Preliminary daily aspirations of the exudate was followed shortly by drainage through an intercostal incision; but the marked dyspnea was not relieved by the thoracotomy. A large pericardial effusion was then found, and the symptoms of heart tamponade necessitated a pericardiostomy which was done through a right sided approach in order to obviate any involvement of the left pleural space. The result of the operation was excellent; the convalescence was disturbed only by temporary edema of the lower half of the body, attributed to pericardial inflammatory exudate about the inferior vena cava. This disappeared spontaneously.

Repeated *x*-ray and electrographic cardiac studies showed no essential change in the cardiac mechanism. At the time of discharge the wounds were healed and the child was in good general condition with apparently no abnormality in the heart or chest. Follow-up observations confirmed this opinion.

ANGINA PECTORIS AND THYROID GLAND

NEW SURGICAL APPROACH

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THE value of surgery of the thyroid gland in the treatment of angina pectoris instigated by the exceptional work and clinical reports of Blumgart in Boston is being very slowly applied to other cases. The reasons are very real why such an operation of apparently very definite therapeutic value in a very large percentage of the human adult population will be reluctantly considered. The two chief drawbacks for depriving the large number of sufferers of angina pectoris from the benefits of relief by this means are (1) the importance is realized of the great danger connected with the operation of total amputation, rather than mere ablation of the thyroid gland, as will be seen later, as a technical problem, rather than in the loss of thyroid secretion. (2) This procedure is still fraught with hazards, chiefly through injury to the recurrent laryngeal nerves, especially in cases where the nerve passes through the substance of the gland, and parathyroid tetany following removal of the parathyroid gland or loss of viability of these bodies from interference with the blood supply which results from the dissection of thyroid tissue in close proximity, and necrosis and absorption later.

These local dangers together with a lengthy operation on a patient weakened by another disease process, as high blood pressure or arteriosclerosis, are very significant and need no further exposure. The regional difficulties are dangerous not only at the time of operation, for it is well known that as long as scar tissue keeps forming after complete removal of the thyroid surgically, injury to the recurrent laryngeal nerves and parathyroid bodies remains a possibility.

These problems are not to be skimmed lightly when one considers such reports as those of McCullagh in 1932 of an incidence of tetany in 1.3 per cent of 11,500 cases in which subtotal thyroidectomy was performed at the Cleveland Clinic, and the posterior part of the gland was carefully preserved. Means and Richardson observed that the frequency of postoperative parathyroid tetany depends roughly on the amount of thyroid tissue removed. And 17 per cent of 73 cases of total ablation of the thyroid gland reported by Gilligan in 1934 showed clinically parathyroid insufficiency. Cutler in a recent report of a series of 54 cases had an immediate postoperative mortality of 4: late unrelated to operation 11; and of 9 complications, 5 were parathyroid tetany and 4 were recurrent laryngeal nerve injuries.

Such reports emphasize the importance one must put on the question, Is the cure or improvement worth the risk? While the skill of the operator is an important factor in preventing injury to these vital structures in this most dangerous anatomical region, it influences very little the reparative, scar forming process that normally follows this operative procedure, Crile warning against dissecting recurrent laryngeal nerves but advising one to leave normal tissue around it to prevent scar injury.

The second drawback to this major procedure is the indirect approach to a disease with these serious consequences the rationale of which is not yet clear. This will be considered only in the light that a less severe procedure will be somewhat more justifiable. For these reasons, a new technique and an operation not so formidable which would accomplish the same

results by removing the secretion of the thyroid gland from the body metabolism in patients with angina pectoris was sought.

Fig. 1.

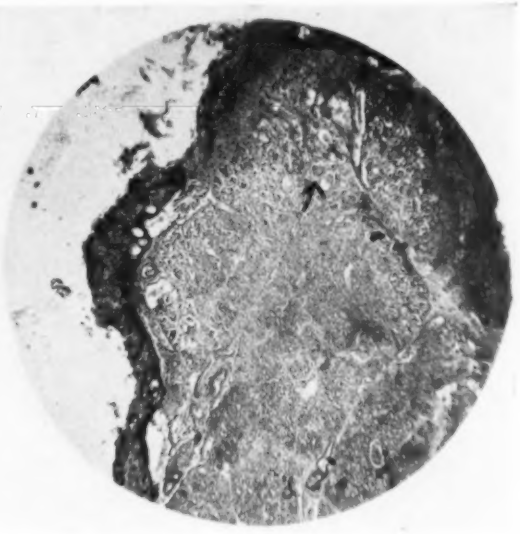

Fig. 2.

It was at this time that the author was working on the blood supply of several of the endocrine glands, chiefly the adrenal, and the effect of the interference of the vascularity on the viability of the granular tissue.

The difficulties that are encountered in maintaining functioning tissue when the blood supply is curtailed, even in small sections of autotransplants is generally well known and was confirmed, both from a physiological viewpoint, and more definitely by histological study of such tissue, under circumstances most favorable for growth. During the past three years of animal experimentation with this problem it has been shown that necrosis and absorption with connective tissue replacement takes place following major obstruction to the original blood supply of the normal gland. And as the vascularity of the thyroid is by far the greatest of any organ in the body and its end arteries without anastomoses, would not its viability be much more quickly effected by a sudden loss of blood supply? And if so, would not the removal of its function in this manner be greatly superior to the total ablation amputation with its risks out of all proportion? With this in mind, animal experimentation was continued to study whatever changes take place in the normal thyroid gland following impedance with its chief sources of blood supply together with the observation for any signs of parathyroid tetany. The microphotographs show a normal gland of the cat under low power magnification, in Figure 1 and in Figure 2 the thyroid gland of a cat five days after ligation of all the blood vessels to it except at its medial attachment, together with separation of the gland from its capsule everywhere except posteriorly, showing the complete necrosis throughout most of the gland; at the part marked by an arrow a small amount of tissue, barely recognizable as thyroid, undergoing necrosis. The same procedure was done in a monkey and a section removed eight days later showed necrosis throughout the tissue but no sign of tetany developed months later.

The safety factor is the prime consideration in this problem, but only so long as the definite result was possible was the procedure to be of value.

Major in his studies on the blood supply of the thyroid had showed that there were no anastomoses in the thyroid gland itself, that only terminal arteries existed in the

glandular tissue: and that whatever anastomatic relationships there were among the thyroid arteries could be found only in

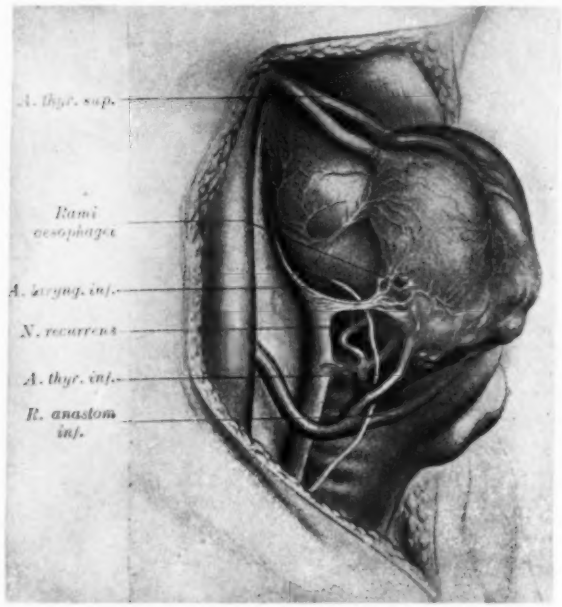

Fig. 3.

the capsule of the gland. Halsted had demonstrated the safety of occlusion of the four thyroid arteries, provided the ligations are done at a safe distance from the parathyroid glandules. With total extirpation of the thyroid it is necessary to ligate closely to these structures, and sometimes even dissect them from the thyroid substance. Enderlen and Hotz in their exacting dissections of the thyroid gland chiefly for its blood supply, have shown, as Figure 3 illustrates, that the terminal part of the inferior arteries receives a branch from the tracheal and esophageal arteries before entering the parathyroid gland as the parathyroid artery. The point of ligature of the superior thyroid artery in this illustration does not apply to the operation here advised, as will be seen. Thus tetany does not supervene providing ligation takes place distal to such anastomoses, an impossibility in the total extirpation as practiced to the present.

These anastomoses are small in a normal subject, but greatly enlarged along with all the other thyroid arteries in the abnormal state.

With the accomplishment of this work a case of angina pectoris was selected which was progressive to the state where the precordial pain was becoming more frequent and severe even while at rest in bed. The prime consideration in the approach of surgery to these patients must be a definite diagnosis, where all known rational medical treatment should have been used for a definite period of time, and found to be of little or no avail in the face of a progressive disease. This case had been hospitalized and medical treatment failed to have any effect. She was told on inquiry as to the treatment of heart disease by surgery of which she had heard, that beneficial results were obtained in some cases but the procedure was a new one. Her condition had become so desperate that she insisted on operation at her own risk.

CASE REPORT

Mrs. M. C., a white female, aged fifty-three years was sent to the hospital with a diagnosis of angina pectoris and hypertension January 7, 1934, for further study and treatment. This diagnosis was confirmed by observation in the hospital where she experienced numerous attacks of precordial pain radiating down the left arm to the finger tips; and by electrocardiographic study. Her stay until her discharge March 24, 1935 was complicated by a partial coronary thrombosis during which attack the temperature rose to 100.8°, pulse 120, white blood cells 10,200; and the radial pulse was reported as thready during the attack. She left the hospital still having frequent typical attacks of pain.

Between the time of leaving the hospital and June, 1935, the attacks increased in severity and frequency while at home with the slightest exertion until finally she was having typical angina pectoris seizures radiating to the left arm and left side of neck. Medication was having very little effect. Her blood pressure was 170/98, pulse 80.

She was operated on June 21, 1935 and from the day after operation through April 21, 1936, ten months later, she has not had a single attack of angina pectoris. The relief was immediate. The operation consisted of doubly

ligating the inferior arteries medial to the common carotid artery and internal jugular vein as it passes behind these and before division takes place, cutting between the ligatures; and the superior thyroid artery as close to its origin from the external carotid as possible. The capsule of the gland was then carefully separated from the entire area except the posteromedian aspect.

She has done most of her household duties, the only complication being a fracture of the lower end of the left radius into the joint, developed through a fall on a slippery floor on September 2, which healed with good function. She also visited a sister in Scranton, Pennsylvania, traveling without any difficulty. She is now living a normal life although warned to avoid excitement and excessive physical exertion. No medication at all has been used. An electrocardiogram November 14 revealed a normal tracing; the blood pressure was 156/92, pulse 80.

DISCUSSION

The result in this case has justified the rationale of the procedure and appears to warrant further use as long as no simpler and more permanent attack on this disease is discovered. The result has been most satisfactory. Whether it has been due to an interruption of the nerve fibers that took place in cutting the thyroid arteries; or whether the localized ischemia of the constantly contracting heart muscle, the latter having been made less sensitive to the adrenal secretion by the absence of thyroid secretion; or whether the mixture of the secretion products of these glands produces this state of altered physiology with its local manifestation is not yet known. Nor has any other explanation as to the mode of action been substantiated. It is not, however, felt that a lowering of the basal metabolism has any direct bearing on angina pectoris; while the metabolism may be at a certain level during a test, that level may vary within wide limits during the day of any patient without noticeable effect. Cutler has reported a case of a myxedematous woman with angina pectoris who had a preoperative basal metabolism rate of −15 to −20 per cent, whose thyroid gland was removed. The basal metabolism rate fell to −20 to −40 per cent. She was relieved of her symptoms even when the basal metabolism rate was raised to her preoperative level by thyroid extract.

That we have here a local manifestation of a generalized altered metabolism, chiefly influenced by the endocrines, and revealed in various regions of the body by similar reactions, may be further emphasized by citing an observation of angina pectoris by Sir James Mackenzie. He told of a patient whose powers of walking was limited for many years by aching legs and feet. On several occasions, after walking considerable distances in spite of the discomfort in his legs, several hours afterward, while resting on a couch was seized with attacks of pain, sometimes of great severity, each attack lasting about a minute and felt in the calf of either leg. These attacks never occurred when he ceased walking long distances, being stopped by pain in the chest. The angina pectoris is but one syndrome of localized vascular muscle spasm, which from repeated attacks may lead to definite pathological findings, to a striking degree approaching the symptoms of altered physiology in other parts. Nevertheless it must be admitted that a definite lacuna in our knowledge still exists which the biochemist must needs bridge in time, until when, relief by whatever means are best and most safely suited, must be employed.

And, as Levine and others, reported, even if thyroidectomy, as an amputation, did not alter in any way the rate of blood flow, basal metabolism rate vital capacity, or other functions, but did relieve the pain, then the operation would still be reliable. And with the distinct advantages of this operation over thyroid amputation, the reason to feel so is greater.

SUMMARY

A new surgical approach to the thyroid gland in the treatment of angina pectoris

is here described with a report of a case.

The operation lacks the dangers of total surgical thyroidectomy as practiced for the same disease.

Animal study, on the thyroid gland alone, was done as a preliminary step.

An attempt has been made to replace a very formidable operation with a comparatively safe one with the same goal in view.

I should like here to express my appreciation to Dr. Fred Griffiths, Professor of Physiology at the University of Buffalo Medical School for permission to use the laboratories for the animal experimentation part of this work and for his kind encouragement.

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MESENTERIC VASCULAR OCCLUSION

REPORT OF CASE OF COMPLETE OCCLUSION OF SUPERIOR MESENTERIC ARTERY WITH INVOLVEMENT OF ENTIRE SMALL INTESTINE*

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WHILE the subject of mesenteric vascular occlusion has been frequently discussed in medical literature since it was first identified by Tiedeman in 1843, it is still an entity of sufficient rarity to warrant the reporting of cases as they are observed, in order that more information might be gathered concerning its nature and behavior, eventually leading to its earlier recognition and more successful treatment. Although many interesting observations concerning the pathogenesis and symptomatology of mesenteric vascular occlusion have appeared in recent publications, the most illuminating resume of this subject has recently been published by Boyce and McFetridge.†

Historical. It was Tiedeman in 1843 who first identified this lesion, and Virchow in 1847 and later in 1854 who accurately described its pathology. It was not before 1863, however, that the first clinical description of this entity was recorded by Kussmaul and Gerhardt, and in 1875 again by Litten. Since then, several series of cases were analysed by various observers, Larson; Jackson, Porter and Quimby; Trotter and others.

Incidence. Owing to the fact that the diagnosis of this disease is most frequently made at operation or autopsy, it is difficult to estimate the frequency of mesenteric vascular occlusion. Some idea can be obtained from reports such as these: Brady found 14 cases by examining the records of the Johns Hopkins Hospital up to 1923, and of this number 3 were recognized at autopsy; Robey was able to collect 51 cases from the records of the Boston City Hospital over a period of 33 years; Boyce and McFetridge were able to collect 13 cases from 30,000 admissions to the New Orleans Charity Hospital over a period of seven and one-half years ending May, 1935, and of this number 4 were recognized at postmortem. Yet, Eisenberg and Schlink, in 1918, reported 4 cases which they had seen in a period of only two months.

Incidence of Age and Sex. The youngest reported case was in an infant one month old, and the oldest ninety years, Jackson, Porter and Quimby. The majority of cases, however, occur between the ages of twenty and sixty years. Men are more frequently affected than women, as indicated in the series of Larson, 26 men to 10 women; Brady, 8 men to 6 women; and Jackson 64 per cent men to 36 per cent women.

Etiology. The exact cause of mesenteric vascular occlusion is not ascertainable in the majority of cases, although it can be stated that, in general, embolus accounts for occlusion of the artery and thrombosis for closure of the vein. Disease of the circulatory system plays the most important role as a contributing cause in this morbid process. Yet the disease, particularly when involving the vein, has been frequently found associated with inflammatory processes of the appendix, 75 per cent according to Polya, and pelvic viscera, with carcinoma of pancreas, fatal cirrhosis, strangulated hernias and a variety of other clinical entities. It has been noted as a not too infrequent postoperative complication

† From the Department of Surgery of the School of Medicine of Louisiana State University and the Charity Hospital of New Orleans, in the International Surgical Digest, 20: 2 (Aug.) 1935. The author has drawn freely from both the text and bibliography of this communication.

* From the Surgical Service, Syndenham Hospital.

of acute appendicitis, vaginoplasties, thyroidectomy and other conditions.

Pathology. Virchow, in 1847, was probably the first to describe the pathology associated with closure of the superior mesenteric artery. Although anatomically the superior mesenteric artery cannot be considered an end artery as demonstrated by Nothnagel, clinically occlusion of its main stem leads to extremely grave consequences. It was Litten who demonstrated that occlusion of the superior mesenteric artery by a thrombus acted physiologically exactly like ligation of any end artery. A statistical survey of the reported cases discloses the interesting factor that the superior mesenteric artery is more frequently involved than is the vein; Jackson, Porter and Quimby, Eisenberg and Schlink report 61 per cent artery to 39 per cent vein; and that in about 90 per cent of the cases it was the superior mesenteric vessels that were occluded, and in only 10 per cent the inferior. As occurs elsewhere in the body, the extent of intestinal involvement depends upon several factors, as for example the size of the branch of the artery affected, the degree of occlusion and the rapidity with which the occlusion occurred. Klein has clearly summarized the possibilities in vascular occlusion of intestinal arteries. He stated three distinct possibilities: (1) the establishment of a competent collateral circulation resulting in no serious results to the intestinal mechanism; (2) intestinal obstruction without infarction due to incomplete occlusion where sufficient blood can reach the involved segment of affected bowel to maintain its nutrition but not enough to enable it to carry on its peristalsis; and finally (3) intestinal infarction varying from the mildest form in which the mucosa alone is involved to a condition where the entire wall of the gut is compromised.

Fig. 1. Photograph demonstrating clot occluding main stem of superior mesenteric artery.

Larson maintained that several incomplete blocks of the lumen of a vascular branch may eventually lead to complete occlusion of that vessel with resultant gangrene. Various explanations have been advanced to explain why occlusion of the superior mesenteric artery acts exactly like the occlusion of an end artery when we know that the intestine is extremely rich in collateral circulation. Boyd's explanation is probably the correct one. He maintains that when a block occurs in the artery, violent spasms of the intestinal musculature are started which cause a further anemia of the involved segment, thus establishing a vicious cycle with resultant gangrene of the involved segment.

In view of the greater susceptibility of the superior mesenteric artery to occlusion, it is obvious why most of the reported cases have involved the jejunum and ileum, the extent of involvement ranging from a few centimeters of intestine to involvement of the entire small intestine, cecum and ascending colon.

The most thorough description of the clinico-pathological findings in cases of mesenteric thrombosis can be found in an

article published by Loop* in which are described the lesions found in 9 cases upon whom laparotomies were performed. According to this author, upon opening the peritoneal cavity one will usually find a certain amount of sticky, more or less transparent amber fluid which at times is blood tinged. The intestine appears soggy and edematous, the serosa is glistening and the color varies from a state of cyanosis to a mottling verging on gangrene. Very little peristalsis is seen. The intestinal lumen, while not distended, is extremely relaxed. The mesentery is thick and doughy and one may at times see or feel the thrombosed vessels through it. Subperitoneal hemorrhages may or may not be seen.

Microscopically, according to Eisenberg and Schlink, the first changes which are detectable are edema and leukocytic infiltration with discrete capillary hemorrhages. Later the vessels and capillaries become enormously dilated. Still later comes the stage of true hemorrhagic infarction with sanguinous extravasation into the intestinal coats, particularly in the submucosa. Necrosis is most marked in the mucosa. Least involved of all is the muscularis.

Symptomatology. The outstanding symptom of mesenteric vascular occlusions, venous or arterial, is abdominal pain which in the fulminating cases is sudden in onset and excruciating in nature, simulating very closely the pain associated with the perforation of a hollow viscus. It may occur in paroxysms or remain constant, and although usually generalized over the entire abdomen, its point of maximum intensity is over the left lower abdomen. Vomiting practically always accompanies the pain, and according to Loop may persist six to eight hours, although in Cases 1 and 3 of Eisenberg and Schlink, vomiting was absent. The vomitus at first consists of gastric contents, but later contains blood, fresh and old. Wilms states that fecal vomiting does not occur. In the fulminating cases shock is frequent. Abdominal distension is usually present and appears early, increasing in severity with the progress of the disease. Peristalsis is absent.

Fever is rare at the onset, although it practically always exists in the later stages of the disease and signifies an accompanying peritonitis. A very striking feature in these cases, as noted by Loop, Dumphy and Zollinger, is the fact that rigidity of the abdominal muscle is slight or entirely lacking in the early stages, but it always exists after peritonitis has been fully developed. The pulse rate in these cases is usually elevated even in the absence of a rise in temperature, and the leucocytes are increased. Some cases show diarrhea with melena, others constipation. Defecation is frequently painful and associated with tenesmus. Borszeky attaches importance to diarrhea followed by constipation.

Diagnosis. A review of the collected cases reveals the fact that rarely has the diagnosis of this disease been made before operation or postmortem. The pain may simulate that associated with any intra-abdominal catastrophe, particularly acute hemorrhagic pancreatitis. The absence of early rigidity clearly differentiates it from ruptured ulcer, while the failure to elicit localized tenderness eliminates acute appendicitis. Unlike cases of intestinal obstruction, visible peristalsis cannot be elicited through the abdominal wall and is absent even with the abdomen open. As clearly demonstrated by Boyce and McFetridge, mesenteric vascular occlusion should be suspected when a patient past middle life, giving a history of having had a cardiac lesion, presents himself complaining of severe abdominal pain with physical findings completely out of accord with the severity of the symptoms, with a temperature normal or even subnormal, but with a pulse rate increasingly rapid and a high leukocytosis. The suspicion should increase in the presence of generalized abdominal tenderness without the presence of rigidity of the abdominal muscles. According to these same authors, a highly suggestive finding is the tendency for the blood pres-

* Loop, R. G. Jour. Am. Med. Assn. (July 30, 1921).

sure to fall. They suggest that repeated blood pressure readings be done in any suspected case.

Treatment. It is scarcely necessary to comment upon the old dictum of operating immediately when a surgical abdomen is suspected, since the best one can hope for in cases of occlusion of the mesenteric vessels is for the physician to realize that he is dealing with a surgical abdomen, and to submit the patient to immediate laparotomy. In the event that a tentative diagnosis of superior mesenteric vascular occlusion is made, it is the concensus of opinion that exploratory laparotomy is indicated in every case. With the abdomen open, the diagnosis is readily made, although the extent of intestinal involvement may be in doubt, particularly in the early stages of the disease. Of all the procedures attempted in this disease, the only one holding forth any hope is enterectomy. Flint, in 1912, published his experiments in the Johns Hopkins Bulletin showing that 50 per cent of small gut could be removed with impunity. The most recent review of this subject was made by Haymond who, after an exhaustive study of the literature, concluded that a patient could withstand a massive resection of 33 per cent of the length of small intestine with good expectancy that the digestive tract would return to normal function, while 50 per cent constituted the upper limit of safety, and resection above 50 per cent would necessarily have poorer results.

Prognosis. Spontaneous cures are extremely rare, as indicated by Klein who found only 8 cases in the literature and added 1 of his own. Occlusion of the mesenteric artery is probably one of the most fatal of all abdominal catastrophes. According to Boyce and McFetridge, the first successful operation for this disease was reported by Elliot in 1894. Thirty years later Klein was able to isolate 23 other successful cases from the literature. Meyer, in 1931, found 29 recoveries of 92 cases, while in Brady's report of 8 surgical cases there were 5 deaths. Boyce and McFetridge had 1 recovery out of 9 patients submitted to operation.

CASE REPORT

Mrs. C. P., housewife, aged forty-eight years, Italian, was brought to the hospital by ambulance on the morning of August 21, 1935. Her chief complaints were nausea, vomiting, obstipation and diffuse abdominal cramps of thirty-six hours duration. Her abdomen felt distended and she stated that she had passed but little urine during the past thirty-six hours.

For the past thirteen years she has had diabetes and was now receiving insulin treatment. Seven years ago her left arm was amputated at the axilla for diabetic gangrene. The remainder of the history was essentially negative.

Physical examination revealed an obese female about forty-eight years of age, appearing acutely ill. The heart and lungs were negative. The blood pressure was 170/76. The left arm had been amputated at the scapula.

The abdomen was uniformly distended but presented no visible peristalsis. Although tenderness was elicited over the entire abdomen, it was most marked over the left upper quadrant. There was no rigidity, but a positive rebound. No masses could be palpated. Rectal and vaginal examinations were negative.

The blood count was unfortunately omitted.

Urinalysis showed sugar 3 per cent; acetone and diacetic acid absent.

The blood sugar was 352 mgm. per 100 c.c. Co^2 63 volume per cent. Temperature 103°F., pulse 126 and respirations 28 per minute.

Owing to the nature of the abdominal findings, a diagnosis was made of an acute surgical abdomen, possibly due to a ruptured gangrenous appendix. The patient was immediately placed upon large doses of insulin and prepared for laparotomy.

Operation. Under intraspinal anesthesia, the abdomen was opened through a long right rectus incision. As soon as the peritoneum was opened, a small amount of brownish, foul smelling fluid escaped. The odor was distinctly cadaveric, which aroused considerable comment amongst those present in the operating room. The small intestine was distended, and deeply cyanotic, but showed no tendency to protrude through the wound. Peristalsis was absent. The caput coli and the greater part of

the ascending colon were deeply mottled and presented the appearance of granite. The mesentery of the small intestine was thick, and of a pale yellowish color. Pulsations were entirely lacking within the vessels of the mesentery. The dilatation and cyanosis extended from the ligament of Treitz down to and including the caput and ascending colon. The descending and transverse colon were not involved. In view of the fact that we knew that we were dealing with a complete closure of the main stem of the superior mesenteric vessels, a massive resection of small bowel was out of the question. As a palliative measure a Witzel enterostomy was performed. The patient, however, succumbed while the abdomen was being closed.

Postmortem Examination. (Dr. A. A. Eisenberg, Pathologist, Sydenham Hospital.)

The body is that of an obese, white, elderly female with a markedly distended abdomen. The left arm is absent. There is a right rectus abdominal incision with a rubber catheter projecting from its lower angle.

An incision was made extending from the suprasternal notch to the symphysis pubis. Upon opening the peritoneal cavity there was an escape of extremely foul odor. The entire gut was distended and blue, and portions of it were of a greenish-black color, being gangrenous. Free fluid was absent in the peritoneal cavity. The thoracic viscera were undisturbed; a gross lesion was not detectable.

The heart was of normal size and shape. The aortic and pulmonary valves were sclerotic but there was no evidence of stenosis or insufficiency. The endocardium was normal but the myocardial muscle was brownish in color and slightly friable.

The pleural surfaces were greyish-red, smooth and shiny and presented no adhesions. The lungs revealed no areas of consolidation. On section a thin, frothy, brownish-red fluid escaped from the lung tissues.

The liver was normal in size and shape. It was brownish in color but slightly paler than normal, its capsule firm and smooth. On section the normal liver architecture was well preserved. There were no gross lesions.

The spleen was normal in size, shape and color. On section the pulp scraped easily, but was of firm consistency. There was no lesion.

The right kidney was normal in size but distorted in shape, the capsule stripping easily except for an area on the lateral margin where it was adherent. At this point there was a depression in the renal mass but presented a yellowish-white color. On section the cortex was thickened, measuring 18 mm. Extending down into the medulla there was a wedge shaped area, yellowish-white in color, having a fibro-fatty consistency.

The left kidney was slightly larger than the right and had a similar infarct to the one found in the right kidney. On section the cortex was markedly thickened, almost obliterating the medulla. No other gross lesions were present.

The entire intestine was tremendously dilated. The stomach contained a thick, yellowish fluid. The small gut from the ligament of Treitz down to and including the cecum was of a greenish-black color, dull and gangrenous. The mesentery showed no abnormalities. On dissecting the mesenteric vessels, a thrombus measuring 11 cm. in length was found in the upper mesenteric artery. It was adherent to the vessel wall and completely occluded the vessel lumen. It was found to extend into the aorta where it was seen to broaden and to completely occlude the osteum, indicating the point of union of the superior mesenteric artery with the aorta. A rubber tube was found in the terminal ilium.

Microscopical. The myocardium shows slight increase in interstitial connective tissue. All vessels show very marked diffuse atheromatous changes and huge calcium deposits, the intima crumbling under the slightest pressure.

The lungs show moderate pulmonary edema. The blood vessels show atheromatous changes.

The liver presents considerable fatty degeneration with a moderate distortion of the hepatic architecture, such as is seen in early cirrhosis.

There is enormous thickening of the capsule and trabeculla of the spleen, with much congestion and a moderate degree of atrophy of the lymphoid tissue.

The right kidney presents an old infarct and an occasional obliterated hyalinized glomerulus and occasionally a tubule showing cloudy swelling is noted. The vessels are markedly thickened due to medial fibrosis and arteriosclerosis. There is no evidence of hyperplastic intimal changes.

The left kidney shows similar changes.

The thrombus in the mesenteric artery is adherent to the intima which shows atheromatous degeneration.

Sections made through one of the diseased intestinal loops shows a typical hemorrhagic and necrotic mucosa with involvement of all layers including the serosa.

Diagnosis. Mesenteric arterial thrombosis.

SUMMARY AND CONCLUSIONS

Although mesenteric vascular occlusion cannot be considered a rare clinical entity, still it is diagnosed clinically so infrequently, that continued comments on this entity in the literature might help make the profession more conscious of its existence and its earlier recognition, leading to more encouraging therapeutic results.

Mesenteric vascular occlusion, involving either the vein or the artery, occurs between the ages of twenty and sixty, and is more than twice as frequent in males as in females. Cardiovascular lesions constitute the most frequent etiological factor in the morbid process. The disease is rarely diagnosed before operation or postmortem. Mesenteric vascular occlusion should be considered when a patient between the ages of twenty and sixty presents himself with severe diffuse colicky abdominal pain, either constant or paroxysmal accompanied by vomiting and a soft, distended abdomen, showing no peristalsis, an absence of fever, in the early stages, and a definite elevation in the pulse rate and leukocyte count. The presence of diarrhea with melena in addition to these symptoms makes the diagnosis extremely probable.

Laparotomy is indicated in every case, and in those cases within the scope of surgical aid, the procedure of choice is enterectomy up to 50 per cent of the length of the small bowel. When the main stem of the superior mesenteric artery is completely occluded, so that the entire small gut and part of the large intestine are involved, the case is hopeless.

Spontaneous cures are extremely rare, and when they do occur, the lesion is undoubtedly a trivial one. In general, it might be stated that occlusion of the superior mesenteric artery is one of the most fatal of all abdominal catastrophies. A cure may be expected when a case is laparotomized early and less than 50 per cent of the small bowel requires resection.

A case is presented by the author, involving a diabetic female patient, aged forty-eight years, with complete closure of the main stem of the superior mesenteric artery by a thrombus and involvement of the entire small intestine, cecum and ascending colon.

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BREAST TUMORS IN CHILDREN*

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MAMMARY neoplasms in the young are of infrequent occurrence and comparatively few articles on the subject appear in the medical literature. In 1908, Jopson, Speese and White[1] attempted to cover the subject quite completely, but found only 19 benign and 9 probably malignant tumors reported as having occurred before the age of sixteen years; and to these they added 2 cases of benign lesions which they had encountered. Of all primary neoplasms of the breast in the young or old, true lipoma and lymphosarcoma are among the rarest. The author has had the opportunity of photographing and studying one of each of these lesions in children, descriptions of which are contained herein with appropriate reference to the literature on the subject, together with a discussion as to the difficulty attending the diagnosis of lymphosarcoma.

BENIGN TUMORS

True benign breast neoplasms are of several different types, only one of which is at all frequent, the well known fibroadenoma, a mixed tumor appearing as a solid or intracystic lesion. Of the 21 benign lesions reported in the mentioned article, 12 were of the fibroadenoma type. Fibroadenomata are known to occur at puberty or early in adolescence, but grow so slowly that they are usually not recognized until the late teens or early twenties. At this age they are frequently observed and many removed but not recorded. The author has studied a 3 cm. fibroadenoma removed from a girl of sixteen years and two smaller ones from a girl of eighteen years, both patients having reached sexual maturity at an early age.

Angiomata of the breast, especially following trauma, are occasionally noted. Jopson et al. report 6 cases in their series, 3 of which were in boys. It is probable that certain cases of so-called breast angiomata are examples of congenital nevi of the deeper layers of the skin rather than of the mammary gland itself. Pure adenoma, melanoma and dermoid cysts of the breast have been reported but are unusual lesions. Fibroma, osteoma, chondroma and teratoid mixed tumors occur, having been discussed by Ewing in his classical work on "Neoplastic Diseases."[2]

LIPOMA

Pure lipoma of the breast proper is one of the rarest tumors, regardless of age, according to Finney,[3] who emphasized the necessity of differentiating between hypertrophy or obesity of the fatty capsule and true lipoma. Kaufmann[4] also regards them as being rare. Bunts[5] records 19 lipomas in a series of 1904 breast tumors, or 1 per cent. Peck and White[6] in a study of 331 breast tumors found 2 lipomas of 136 benign growths. Smith and Marks[7] found no lipoma in 201 cases of benign breast tumors seen over a period of fifty-three years in one hospital. Bland-Sutton in his textbook[8] does not mention lipoma of the breast. Bloodgood[9] in 1908 stated that he had observed only 3 cases. Lipomas arising in the axilla and around the shoulder are occasionally seen. A few are reported as having been in the subpectoral space as in the case of Torraca,[10] who removed one

* Read before Chicago Pathological Society, Nov. 11, 1935. From Loyola University, School of Medicine, and Pathological Laboratory, St. Bernard's Hospital.

weighing three pounds from a seventy-three year old male.

True lipomas of the breast in children are extremely rare. Martin's case[11] is reported as a lipofibromyoma, a mixed tumor, weighing 33.3 pounds, having been removed from a sixteen year old colored girl. Atkins[12] reported a fatty tumor weighing 25 pounds removed from a sixteen year old Indian girl.

The following is a report of a pure lipoma of the breast in a four and one-half year old boy.

W. C. G., aged four and one-half years, was admitted in 1931, because of a gradual swelling of at least six months' duration in the left breast. Examination showed a healthy normal boy with a soft rounded diffuse non-tender swelling below the left nipple. (Fig. 1.) The other breast was normal and there were no nodules elsewhere on the body. The mass was removed under general anesthesia. The wound healed well and there has been no evidence of recurrence in over four years. The pathological report was as follows. (Fig. 2.) "Specimen consists of a well-demarcated round mass of soft pale yellow fat, 5 cm. in diameter and 2 cm. thick at its center. It is partially surrounded by a very thin delicate fibrous capsule and is overlaid by a narrow elliptical piece of non-adherent skin, including the nipple, which is small and partially inverted. Sections show the mass to be made up of pure fat with an occasional delicate fibrous septa. No glandular tissue is seen. Diagnosis: Lipoma of the breast."

MALIGNANT TUMORS

Malignant neoplasms in children and young adults are comparatively uncommon but are by no means rare. Bulkley[13] states that in the United States in 1918 there were 65,340 deaths from malignant disease. Of these, 593, or 0.9 per cent, were children fourteen years of age or under. Of that group there were 381 with sarcoma, 203 with carcinoma, and 9 with hypernephroma.

CARCINOMA

Malignant tumors of the breast in children have been reported on several occasions. A critical analysis of these indicates that many of them have not been diagnosed microscopically. In others the diagnosis is highly questionable. Phillip[14] collected 390 cases of various carcinomata reported in children, of which only 87, or 22.3 per cent, withstood critical analysis. He did not find a single case of mammary cancer under fifteen years. Lee[15] studied 2663 patients with cancer of the breast over a twelve year period. Two women aged twenty-two years were the youngest in the group. The United States census for 1914 reports 5406 deaths from mammary cancer, 4 of which were in the fifteen to nineteen year old group. Among the reported cases of carcinoma of the breast in children, Jopson et al.[1] in 1908 found only 3, girls eight, thirteen and sixteen years of age, all of which were questionable, having occurred many years previously.

Luttinger[16] reports a probable cancer of the breast in a girl of thirteen years, but the diagnosis was not confirmed. He quotes Chauvel and Renaud as reporting a case similar to his in a girl of fifteen years.

The earliest reported case which seems authentic was described by Blodgett[17] in 1897, a boy of twelve years who remained well for at least five years following the removal of a breast tumor, which presented the typical structure of carcinoma on microscopic examination. In 1908 Thompson[18] described an adenocarcinoma removed from the breast of an eleven year old girl. Levings[19] in 1917 reported a glandular carcinoma in a girl of twelve years. He records 2 other cases, that of Fowler in a girl of seventeen and one-half years, and Bryan in a boy of fourteen and one-half years following trauma. Kaufmann[4] mentions one at seventeen years and Hill[20] at nineteen years. More recently, in 1932, Carnett, Widemann and Howell[21] reported the case of a fourteen year old girl on whom a mammectomy was performed for a carcinoma, apparently of the alveolar type, describing her as "the youngest patient with breast cancer of whom we know." Their article is illustrated

with a photomicrograph showing a picture very similar to that of the tumor in the case reported here.

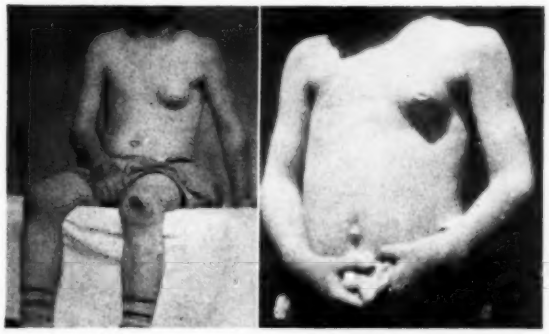

Fig. 1A. Fig. 1B.

Fig. 1. Lipoma of breast in boy aged four and one-half years.

SARCOMA

Sarcoma of the breast in children is described in several articles appearing in medical literature, but in many of these the histological data is omitted, and the accuracy of the diagnosis is open to question. Jopson et al.[1] recorded 6 cases between the ages of four months and fourteen years, one of which, at least, was not examined microscopically; the others are also questionable. Rouffart[22] in 1905 reported a large celled fibrosarcoma in both breasts and both axillary glands in a girl of fourteen years. In 1910 Lyppens[23] described the removal of a fibrosarcoma from the breast of a girl of sixteen years, with local recurrence in three months. Fox[24] in a series of 60 cases of mammary sarcoma noted one occurring at the age of thirteen years. Incidentally, in this series only 7 of the 60 cases were of the lymphosarcoma type, the majority, 70 per cent, being fibrosarcoma.

The following case report is that of a fifteen year old girl who died following the removal of a lymphosarcoma of the breast.

Miss V. B., aged fifteen years, was admitted to St. Bernard's Hospital in 1933. She was a nervous, easily excitable girl of defective mentality. Her history was given by the mother, who three months before had noticed a small lump the size of a large hazel nut below the nipple in the patient's right breast. This steadily enlarged until it was the size of an apple, causing a dull aching pain. During the last month a lump in the medial half of the left breast gradually developed to the size of a

Fig. 2. Pure lipoma removed from breast of boy shown in Fig. 1.

hen's egg. Three years before the girl had fallen on a fence, striking the right breast and cutting the nipple. There was no other history of injury. She had had measles, whooping cough and chorea as a young child and had always been "jerky, excitable, thin and sallow." At the age of six or seven years a slight enlargement of the thyroid was noticed but no metabolic studies were made. Tonsillectomy was performed at ten years.

The family history was negative for cancer, diabetes, tuberculosis, nephritis or nervous disorders. Her mother had two pregnancies. The patient was the first child and was delivered by forceps after a very difficult labor. The second pregnancy was terminated by Caesarean Section in 1932. The child, a boy, died at six weeks and an autopsy performed by the author revealed a congenital heart lesion and generalized anascara but no evidence of syphilis or neoplasm.

Physical examination revealed an anemic appearing, somewhat undernourished white girl, who resisted examination, and appeared to have the mentality of a child of six. The right breast was enlarged by a hard, round, fixed, slightly lobulated tumor, over which the skin was freely moveable. A small, softer, but also fixed mass was palpable in the right axilla, and another fixed mass, the size of a hen's egg, in the upper medial quadrant of the left breast. There was a moderate, soft, sym-

metrical enlargement of the thyroid gland. Examination of the chest revealed no lung pathology. The heart was rapid, but appeared normal in size, and no murmurs were heard. There were no other significant physical findings.

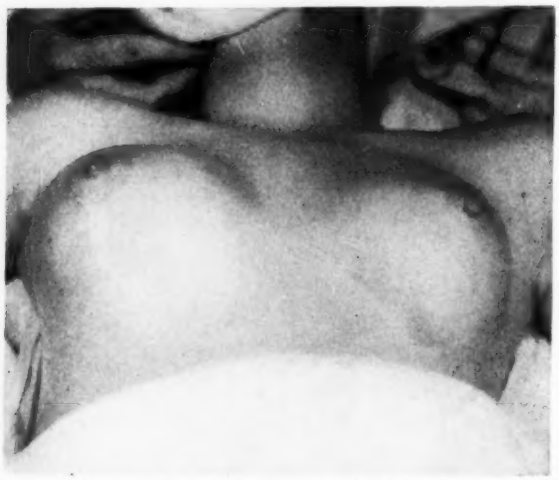

Fig. 3. Large lymphosarcoma of right breast with smaller nodule in left breast of girl aged fifteen years.

Laboratory examination revealed hemoglobin 80 per cent, red blood cells 4,200,000, and white blood cells 13,000, with a normal differential count. The urine was normal. Her mental condition prevented metabolic studies and unfortunately roentgenograms of the chest were not made. The symptoms and physical findings did not suggest any mediastinal pathology.

The girl objected to a photograph, which had to be made under general anaesthesia prior to the operation. (Fig. 3.)

The diagnosis was made of a malignant tumor of the right breast, and mammectomy was deemed advisable even though it would give only a temporary check to the tumor growth. A radical right mammectomy was performed, removing the breast and axillary mass. Her postoperative mental condition was not good and four days after operation she was discharged to her home where she died two weeks later. Permission for autopsy could not be obtained.

The pathological report of the specimen (Fig. 4) is as follows:

"*Gross Examination.* This specimen weighs 536 grams, and consists of a large tumor of the breast, partially covered by an elliptical piece of skin, including the nipple, and measuring 17 by 7 cm. The areola measures 3.5 by 2.5 cm., and is not attached to the underlying tumor. The main tumor mass measures 9 by 7 by 6 cm.,

Fig. 4. Surfaces made by sectioning surgical specimen of lymphosarcoma of right breast shown in Fig. 3.

is firm, well defined with rounded edges, homogeneous, with distinct lobulations, glistening on surfaces freshly made by cutting, and grey, with red-brown peripheral mottling. Two smaller, separate masses, are softer, of almost brain-like color and consistency, are not as well demarcated, and measure 3 and 2.5 cm. in diameter respectively. The remainder of the breast is firm, fatty and fibrous tissue. The small mass of axillary fat contains a broken-up, soft, grey nodule of tumor tissue, 3 cm. in diameter.

"*Microscopic Examination.* Sections made from various portions of the main tumor mass and other nodules show essentially the same picture. There is a diffuse overgrowth of small round undifferentiated cells. These are closely packed, fairly uniform in size and shape, and vary only slightly in the chromatin content. Occasionally a flattened or polyhedral tumor cell is noted, but in no field is there seen the typical formation of the spindle cell of fibrosarcoma. Typical mitotic figures are rarely seen. In places the cells seem to lie in poorly outlined alveoli, but these alveoli are not surrounded by definite strands of stroma. Elsewhere the cells are found in irregular masses and infiltrating strands, especially at the periphery, where a hyalinized stroma appears. This stroma is evidently the connective tissue of the breast, which is invaded by tumor,

rather than a part of the neoplastic process. Scattered through the tumor are occasional remnants of breast glands and ducts, lined by a single epithelial layer with desquamation into the lumen. These glands are surrounded and compressed by the tumor tissue and there is no evidence that they have any part in the neoplastic growth. (Fig. 5.) Careful study under higher power shows a definite reticulum between the round cells of the tumor. This is seen as fine thread-like fibres with typical 'reticulum' cells, which are larger and more irregular in shape, and distinctly paler than the other tumor cells.

"The homogeneous softness and color of this tumor suggest a neoplasm of mesoblastic origin. The uniformity of the small round tumor cells, their undifferentiated manner of growth, the surrounding of ducts and glands without obvious invasion, and the presence of definite reticulum cells speak for a sarcoma of the lymphogenous type. Diagnosis: Lymphosarcoma of the breast."

DISCUSSION

The term "round-cell" sarcoma is widely used and apparently is accepted as accurate by the majority of pathologists. There can be no doubt, however, that many and perhaps most of the neoplasms so diagnosed are not round cell sarcoma but rapidly growing anaplastic small cell carcinoma. Ewing[2] recognizes round cell sarcoma originating in the lymphoid and glial tissues and in the bone marrow, but doubts their origin from other mesoblastic tissues. Perfect fixation and a study of various portions of a tumor often show that the so-called round cells are really spindle shaped or polyhedral cells and should be so classified. Round cell tumors arising from soft tissues of the body are, with rare exception, either lymphosarcoma or small cell carcinoma. A differential diagnosis is often extremely difficult and sometimes impossible, as both carcinoma and sarcoma cells may show similar and extreme variations in morphology, diagnostic of malignant neoplasm but not specific for any particular tumor.

Virchow[25] believed that an intercellular stroma must be identified to classify a round cell tumor as sarcomatous. White[26] noted a fine intercellular reticulum very constantly in sarcoma, but could not detect

Fig. 5. Lymphosarcoma of the breast. High power photomicrograph showing two breast ducts surrounded by the uniformly small, round, dark staining tumor cells. Scattered among the latter can be seen several of the paler "reticulum" cells.

it in carcinoma. Seelig[27] also described the reticulum of sarcoma. Thus for many years the presence of reticulum cells has been nearly generally regarded as a criterion for the diagnosis of sarcoma, but Ewing[2] believes that the diagnostic significance of this feature of sarcoma has been over emphasized. Certain round cell tumors apparently composed of lymphocytes or their precursors, lymphoblasts, are described under the terms of "lymphocytoma" and "lymphoblastoma."[28] These may lack the reticulated stroma of small round cell sarcoma arising from supporting connective tissue and internal organs.

This brief discussion will serve to explain the reasons for the diagnosis of lymphosarcoma in the patient here re-

ported. Others might prefer to describe it simply as a round cell sarcoma, even an example of a highly anaplastic, rapidly growing form of carcinoma. It is unfortunate that this chapter of oncology is still in the same state of confusion in which it was in 1894 when Williams[29] was unable to collect sufficient data to write the history of pure mammary sarcoma. Ewing[30] states: "When one excludes from the group of mammary sarcomas the adenosarcomas, the malignant forms of mixed tumors containing cartilage, bone, mucoid, or fat tissue, and certain malignant round, spindle and giant cell alveolar pseudosarcomas, which are really atypical carcinomas, there is little remaining of a once formidable group of mammary neoplasms."

CONCLUSION

Tumors of the breast are seldom seen in children.

Lipoma and lymphosarcoma are two of the rarest mammary neoplasms at any age.

Herewith are reported a breast lipoma in a four-year old boy and a lymphosarcoma of the breast in a fifteen-year old girl.

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GAS GANGRENE INFECTIONS FOLLOWING APPENDECTOMY*

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WOUND infections produced by anaerobic gas forming bacteria are comparatively rare following abdominal operations. They may occur after any type of laparotomy but are most frequently seen after surgical procedures performed for the relief of intestinal obstruction.

In 1932 Millar[1] found reports in the literature of 48 cases of such gas gangrene infection, and since that time 4 have been added. Of these 52 cases only 10 have followed appendectomy: 8 were presented by Millar[1] in his report; one was reported in 1933 by Traver;[2] and one in 1934 by Orr.[3]

Two cases of gas gangrene infection following appendectomy occurred on the Surgical Service of the Fifth Avenue Hospital within the last two years. The reports of these two cases follow.

CASE REPORTS

CASE 1. Mr. R. C., age thirty-nine years, was admitted July 20, 1933, complaining of pain in the right lower quadrant since July 16, which had a sudden onset associated with nausea. The pain was at first in the epigastric region but later settled in the right lower quadrant and remained there. On July 18 he began to have frequent bowel movements, having five evacuations on the following day and two on the morning before admission. He had noticed a feeling of discomfort on urination but there was no actual pain, frequency or hematuria.

Examination showed a white male who appeared to be acutely ill. There was a drawn look to the face and a distinct palor. Abdominal examination presented exquisite tenderness over McBurney's point. There was a suggestive, small, round mass about 3 to 4 centimeters in diameter over the region of the cecum. There was no rigidity or spasm of the muscles of the anterior abdominal wall and no skin hyperasthesia. Rectal examination showed some tenderness to the right; no palpable masses; prostate normal.

Laboratory Findings. The urine showed a very faint trace of albumin, and occasional white blood cell.

The white blood count showed 10,300; polymorphonuclear, 67 per cent; lymphocytes, 32 per cent; eosinophiles, 1 per cent; nonlobulated, 36 per cent.

Diagnosis. Active progressive appendicitis with localized peritonitis.

Operation, July 20, 1933. A McBurney incision was made. The cecum was freed by separating numerous dense adhesions and a small abscess which was well walled off was unavoidably opened. Only about one inch of gangrenous appendiceal stump remained. Because of the difficulty of exposure due to its retrocecal position, it was not deemed advisable to attempt to remove it. A cigarette drain was inserted into the region of the abscess cavity emerging through a suprapubic counter incision. The peritoneum was closed. The wound was packed with vaseline gauze.

For three days following operation the patient's condition remained satisfactory, his temperature and pulse both receding normally. On the third p.a.d.* his temperature was 100° and his pulse 80. On the morning of the fourth p.a.d. his pulse suddenly jumped to 140 and his temperature rose to 102.8. Examination showed a pulse weak, regular and rate of 150, blood pressure 94/72. Respirations were not labored. There was a thin, warm perspiration on his face and neck. He complained of severe left lower abdominal pain which was cramp like. The entire abdomen was slightly distended and

* The term p.a.d. is used to denote post-admission day. This is done to avoid confusion. Because of the number of operations this patient had performed the usual term p.o.d. (postoperative day) would become ambiguous.

* From the Surgical Service of the Fifth Avenue Hospital, New York City.

there was a particularly marked distention of the upper portion. He was passing gas by rectum and had had a good bowel evacuation the previous evening following an enema. There was no vomiting but considerable nausea and belching of gas. On exploration of the McBurney incision, a quantity of dark, reddish-brown, foul smelling discharge was obtained. A soft rubber catheter was inserted into the peritoneal cavity in this area. From the appearance and odor of the discharge and the dark appearance of the muscles, Bacillus Welchi infection was suspected. Anaerobic cultures and smears were taken. The smears showed the presence of large Gram-positive encapsulated bacilli resembling Cl. Welchii. Cultures were reported the following day as positive for Cl. Welchii and streptococci.

Treatment. The stomach was lavaged by means of the Levin tube which was left in place for irrigating every hour; 2,000 c.c. of 5 per cent glucose-saline were given by hypodermoclysis and 500 c.c. of 2 per cent saline intravenously and the patient morphinized to prevent pain and shock.

One ampule of concentrated polyvalent gas-gangrene antitoxin was given intravenously.

From the fifth to seventh p.a.d. the pulse remained about 150 dropping down to 120. The temperature ranged from 103° to 104°. Supportive treatment was continued.

On the eighth p.a.d. the patient had four watery stools during the previous night. Examination showed the presence of a palpable mass extending from the pelvis to three fingers breadths below the umbilicus and from the midline to the left for about 10 cm.

Diagnosis. Secondary intraperitoneal abscess.

Operation. An incision was made to the left of the midline. On opening the peritoneum there was a gush of purulent, foul smelling pus; about 1000 c.c. was removed. A large walled off abscess cavity was found extending down practically to the bladder. Two rubber tube drains were inserted. The wound was packed with vaseline gauze. Immediately following operation a blood transfusion of 500 c.c. was given.

Cultures from the fluid taken at operation showed the presence of streptococci, but no Cl. Welchii or other gas forming bacteria.

The supportive treatment was continued. The pulse ranged between 90 and 110 and the temperature between 102° and 103°. Thirteenth p.a.d.—the patient complained of some pain in the right upper quadrant. Examination of the chest showed flatness on the right side up to the angle of the scapula with absence of voice and breath sounds. There was tenderness on compression of the lower ribs. *X*-ray examination showed the two diaphragms on a level and within normal limits. There was no gas beneath the diaphragm and no evidence of subdiaphragmatic abscess. There was some congestion of the right base but no consolidation.

Fifteenth p.a.d.—the breathing was more shallow. The chest signs had remained the same as at the time of the previous examination except that large ronchi could be heard over the entire chest. *X*-ray examination showed no change from the previous films. The differential diagnosis rested between a process above the diaphragm or below it. In view of the *x*-rays and the history a *diagnosis* of subdiaphragmatic abscess was made.

Operation. An incision was made along the twelfth rib on the right side and three-quarters of the rib removed subperiosteally. A transverse incision was made into the retroperitoneal space. Many dense adhesions were broken down. An aspirating needle was inserted and pus was found. This needle was followed and a large abscess cavity was opened over the dome of the liver. The cavity was evacuated and drained. The patient left the table in good condition.

During the remainder of the day the patient's condition again became critical. The temperature jumped to 104°, pulse 144, respiration 18, blood pressure 92/62.

Sixteenth p.a.d.—morning temperature was 103.6°, the pulse was a great deal stronger and the rate had dropped to 100. The patient was given a blood transfusion of 500 c.c. He returned from the transfusion room at 1:50 P.M.

2:15 P.M.. Severe chill; p. 152; r. 40
2:30 P.M.. Chill stopped; p. 112; r. 36; patient drowsy
3:15 P.M.. Severe chill; t. 106; p. 160; r. 40
3:45 P.M.. Perspiring profusely; t. 105; p. 160; r. 32
4:15 P.M.. T, 103; p. 120; r. 24
5:45 P.M.. T, 101.2; p. 112; r. 24

From the sixteenth to twenty-first p.a.d.—supportive treatment was continued consisting of hypodermoclyses, intravenous infusions, morphine, colonic irrigations, etc. The temperature ranged from 104° to 102° gradually receding. All during the period there was a great deal of discharge from all incisions and the dressings had to be changed very

frequently. His general condition, however, improved steadily. With each hypodermoclysis of 5 per cent glucose-saline, five units of insulin were given. At the site of the McBurney incision a fecal fistula developed. A Carleton suction apparatus was placed in this incision and was run continuously in order to keep the wound as clean as possible. On the twenty-first p.a.d. the patient's temperature was lower and he seemed better but he gave a history of rather excessive cough during the last forty-eight hours. On examination the right chest was dull from the spine of the scapula down, slightly distant voice and breath sounds over this area, no bronchial breathing or rales. On the left side there was an area of dullness at the apex of the scapula, many coarse moist rales and an area about 2 cm. in diameter of bronchial breathing. *X*-ray showed a lung and pleural reaction throughout the right side of the chest, chiefly pleural, which had increased considerably since the last examination. There was no appreciable amount of fluid. There was an infiltrative process in the base of the left lung of a pneumonic type. Both diaphragms were lower at this time than previously, the right by one interspace.

From the twenty-second to thirtieth p.a.d., at no time did the temperature reach normal. In the mornings it came down to 101° and in the afternoons it spiked to 102°. All the wounds were discharging less and the fecal fistula was almost closed. His cough persisted however, and on the twenty-fifth p.a.d. the *x*-ray was repeated. It was reported that the process in the right chest was a little more advanced but that it was limited to the pleura and that the lung markings could be seen through the increased density produced by the pleural reaction. There was only slight increased density over the last film and there was no appreciable amount of fluid present.

On the thirtieth p.a.d. on physical examination the dullness and distant voice and breath sounds persisted. The *x*-ray films showed a slight increase in density in the right chest, indicating an increase in the pleural process and that there was a thin layer of fluid over the surface of the right lung. The lung markings were still clearly visible through this increased density. The amount of exudate was not very great. The left lung was clear. In spite of the fact that the *x*-ray showed no localized reaction and a relatively minor pleural reaction, thoracentesis was done and 80 c.c. of thick, foul smelling pus was obtained.

Diagnosis. Empyema, right.

Operation. Under local anesthesia an incision was made in the posterior axillary line over the ninth and tenth ribs and about 3 inches of these ribs removed subperiosteally. An aspirating needle was inserted and pus was found. An incision was made through the pleura over the lower edge of the upper lobe. A large amount of pus was evacuated and the cavity was packed with vasoline gauze. Cultures taken of this fluid showed the presence of streptococci; no anaerobic gas forming bacteria.

The temperature gradually dropped after this operation. The patient began to pick up rapidly and gained considerable weight. The packing in the chest was changed under gas anesthesia four days later and was removed from the chest on the thirty-ninth p.a.d.

On the fortieth p.a.d. the patient was allowed out of bed. On the forty-fourth p.a.d. a blood transfusion was given of 500 c.c. following which there was no reaction. On the fifty-third p.a.d. the patient had a chill and the temperature rose to 103°. The chest wound was probed and a pocket of pus opened and drained. The temperature immediately fell to normal and remained there. *X*-ray examination on the fifty-fifth p.a.d. showed a slight amount of air in the pleural cavity overlying the lung but the expansion of the lung was practically normal. There was slight thickening of the pleura but no pocketing. The patient continued to improve and was discharged on September 18, 1933, his sixtieth p.a.d.

All the wounds were completely healed and the patient was discharged from the Out Patient Department two weeks later.

He has returned for follow-up examination three times since his discharge. The last visit was in September, 1935 at which time all the wounds were well healed. There were no hernias in any of the abdominal wounds. The patient had no complaints except a tendency to constipation which he could readily regulate by diet and the taking of mineral oil. He is working every day and considers himself in the best of health.

In this case the gas gangrene infection was limited to the wound area. Although the patient's general resistance was poor,

supportive measures plus one therapeutic injection of antitoxin were sufficient to enable him to overcome the infection. None of the subsequent complications were due to gas gangrene bacteria.

Case II. Mr. J. van C., aged thirty-two years, was admitted May 29, 1935, complaining of severe pain in both lower quadrants. The patient became ill three days previously with pain in the abdomen which was not localized but was felt mostly in both lower quadrants. He took a violent cathartic the following day. To overcome the pain and partially because the next day was his birthday, he took considerable quantities of alcoholic beverages. Following the birthday party, which began the night before admission and ended on the morning of admission, the pain increased as the effects of the alcohol decreased. At this time the pain was colicy in nature, limited to the lower abdomen, extremely severe and was accompanied by nausea and vomiting. Movements of any kind became extremely painful and the patient was forced to lie in bed with the knees drawn up and not moving from one position. He was brought to the hospital by ambulance.

Physical examination showed a moderately obese young man lying quietly in bed, appearing acutely ill and in considerable pain. Temperature on admission was 101.6°, the pulse was 80 and respirations 20. He was extremely apprehensive and had made up his mind that he was not going to recover. The skin was warm and moist. The abdomen was slightly distended and held rigidly, not moving on respiration. On light palpation there was skin hyperasthesia all over the lower abdomen. On deep palpation the lower abdomen was absolutely rigid and there was marked tenderness in both lower quadrants and over the symphysis pubis, but greater over the symphysis than on either side. There was marked rebound tenderness all over the lower abdomen. No costovertebral tenderness was palpated.

Laboratory Reports. Urinalysis showed a very faint trace of albumin with a few red blood cells, occasional single and clumped white cells and a few epithelial cells.

White blood count of 8000 cells with polymorphonuclears, 81 per cent; lymphocytes, 17 per cent; monocytes, 2 per cent; nonlobulated, 43 per cent.

The patient was given morphine ¼ grain. One-half an hour later examination showed a marked diminution in the tenderness in the left lower quadrant and over the symphysis, with maximum tenderness directly over McBurney's point. Rectal examination showed tenderness on both sides, but more pronounced on the right. No masses were felt by rectum. The prostate was normal.

Diagnosis. Active progressive appendicitis with peritonitis.

Operation. On opening the abdomen through a McBurney incision, a moderately increased amount of clear fluid was found in the peritoneal cavity. On placing the finger in the right lumbar gutter a considerable amount of pus was liberated and welled up into the wound. This pus was evacuated with the sucker and a moderate number of freshly formed adhesions were separated and the appendix was readily brought into the wound. It was found to be acutely inflamed, particularly at the tip and up to the midportion where a gangrenous area almost completely separated it from the proximal portion. The base of the appendix was clamped and tied; the appendix removed and the stump carbolized. A stab wound was placed into the right lumbar gutter from the right flank and a rolled rubber dam drain placed into and along the gutter. The peritoneum was closed and the wound was packed with vaseline gauze strips.

Pathological Report. The specimen was received in the laboratory in two pieces. *Diagnosis.* Acute gangrenous appendicitis and periappendicitis.

For three days the patient ran a perfectly normal postoperative course considering the operative findings. The temperature ranged below 100° all the time and the pulse never went above 100. The midnight temperature on the third p.a.d. rose to 101°, and the following morning it was 102°. At the same time the pulse rate jumped up to 120. At this time a moderate distention developed and the patient became nauseated and vomited. Relief was obtained by giving a milk and molasses enema. That evening the temperature went down to 101.4°, after reaching a high for the day of 103°.

On the fourth p.a.d. the temperature range remained around 101°. The pulse rate, however, continued high and reached a peak of 140 toward evening. Nausea and vomiting con-

tinued. At the same time he developed a severe attack of lower left side abdominal pain. Examination showed a moderate distention of the lower abdomen and considerable tenderness in the left lower quadrant. Large quantities of gas were being passed by rectum. It was thought that the patient was developing a left sided abscess in the peritoneal cavity. However, no obstruction was present. The dressing was changed. There was only a moderate amount of discharge. The wound was packed using plain gauze soaked in azochloramid solution in oil. At 5:30 P.M. the patient vomited a large quantity of black, foul smelling fluid. He was bathed in perspiration and his general appearance was that of a very sick man. Since morning there had been very little gas expelled by rectum and he still complained of a good deal of pain in the left lower quadrant. His stomach was immediately lavaged by means of the Levin tube which was left in situ for irrigation every hour. A clysis of 1000 c.c. of 5 per cent glucose in saline was started. At 7:00 P.M. he passed a small amount of reddish colored urine. At 9:00 P.M. he had an extremely copious movement from the bowel, evacuating a large amount of fluid and flatus, following which he was greatly relieved. A consultation was held at 10:00 P.M. at which time the dressing was removed. The discharge had increased tremendously, so that there was a steady drip from the drain in the stab wound. This discharge was dark, reddish-brown in color and had a mousy, stale meat smell which was very offensive. The same type of discharge was found in the McBurney incision and one could see small bubbles of gas rising in it. Smears and cultures were taken and a tentative diagnosis of gas bacillus infection was made. One therapeutic intravenous injection of mixed gas-gangrene antitoxin (Lederle) was given immediately. Morphinization treatment was begun.

On the fifth p.a.d. the smear showed Gram positive encapsulated bacilli, morphologically resembling Clostridium Welchii; on culture Eschericia coli predominated, but streptococcus was also present. Clostridium Welchii and probably other species of clostridium were present.

The patient remained extremely weak; 2000 c.c. of glucose-saline were given by hypodermoclysis and two more therapeutic doses of gas-gangrene antitoxin were given intravenously. The patient's general condition improved slightly and he was able to take and retain some fluids by mouth. The temperature remained at 102°, but the pulse rate varied from 120 to 140 and was weak and easily compressible.

On the sixth p.a.d. the patient was still very weak in the morning and complained of nausea and considerable left lower quadrant pain. There was a moderate suppression of urine. A fourth injection of gas-gangrene antitoxin was given intravenously, and he also received 2000 c.c. of glucose-saline by hypodermoclysis. A blood transfusion of 350 c.c. was given in the afternoon. Following transfusion the patient picked up considerably. His temperature dropped to 100, and his pulse volume was greatly improved in spite of the fact that the rate continued high.

The seventh p.a.d. an intravenous of glucose-saline was started in the morning and allowed to drip slowly most of the day. The patient in general was improved. He could take some fluid by mouth, the quantity of urine output increased and for the first time he seemed to be progressing favorably. The patient's mental condition improved also and he began to believe that he was going to recover. A donor was obtained for a transfusion to be given the following day. The temperature remained about 100°, the pulse stayed regular and of fair volume, the rate being about 110. The amount of discharge from the wounds was greatly lessened and the offensive odor had greatly decreased.

On the eighth p.a.d., during the night the pulse rate rose rapidly, and the volume decreased with the increased rate. Hiccough, vomiting and rapid respirations began. The patient sank rapidly as though overwhelmed by a severe toxemia, he became unconscious and died. Autopsy could not be obtained.

SUMMARY OF CASE II

In this case the gas-gangrene infection produced extreme toxemic symptoms. Though not proved by autopsy this patient undoubtedly had a gas-gangrene peritonitis rather than a limited wound infection.

There have now been 12 cases reported of gas-gangrene infection of abdominal wound following appendectomy. In 9 of the cases at the time of operation the appendix

was gangrenous, perforated and small abscesses had formed. Shearer[4] reported one case in which the appendix had become gangrenous but had not perforated. Eckhoff[5] reported one case in which there was an active progressive inflammatory process, but there were no gangrenous areas in the appendix and one case in which operation was performed for chronic appendicitis. The latter patient had a rather stormy postoperative course and developed gas-gangrene infection of his wound on his twentieth postoperative day.

It has long been an established fact that the Bacillus Welchii is normally present in the intestinal tract. It is reported by Jennings[6] that in 90 per cent of appendices removed at operation, Bacillus Welchii has been found by cultures made from the contents, to be inhabitant of the appendiceal lumen. This author has also recovered Bacillus Welchii from the muscular wall of an inflamed appendix, as well as from the lumen content, and also from localized collections of pus. In 40 cases in which cultures were taken from free fluid in the pelvis or general peritoneal cavity, Bacillus Welchii was found 10 times.

In a report on the bacteriology of the peritoneal cavity by Roberts and Johnson[7] it is stated that anaerobic spore bearing rods were recovered at the time of operation in 14 cases of appendicitis and in 12 of these it is definitely stated in the reports of both surgeon and pathologist that the organ was gangrenous. Once since that report anaerobic cultures have been found to be positive for Clostridium Welchii in a similar case. In none of these cases did there subsequently develop any sign of gas-gangrene infection of the wounds.

In all cases of appendicitis in which there is a gangrenous process present at the time of operation, the possibility of the later development of a gas-bacillus infection of the wound should be borne in mind. This is particularly true if a localized collection of pus or a general peritonitis has developed. Butler[8] states that "suspicion of such a complication should be aroused by a sudden increase in the pulse rate, with or without a rise in temperature." This was well demonstrated in these 2 cases, and was accompanied by a marked diminution in blood pressure. There may be a great deal of pain felt at the site of operation but this did not occur in either of the cases reported here, so it is not a point on which to rely. These symptoms and signs usually occur on the second or third postoperative day. The signs of an overwhelming toxemia enter at the same time with nausea, vomiting, extreme weakness, profuse perspiration and the anxiety of impending death. Examination of the wound may show a coppery discoloration of the skin and the characteristic profuse discharge of a dark reddish-brown color with a sweet, pungent, nauseating, mousy odor. Small bubbles of gas can frequently be seen rising in the fluid which bathes the wound. Any exposed muscle tissue will appear dark and lifeless and will not contract on stimulation. Smears from this discharge show the large encapsulated Gram-positive bacilli of Welch, as well as other bacteria. Anaerobic cultures should be taken. Once this diagnosis has been made or suspected, treatment should be instituted immediately and not delayed for any laboratory confirmation.

All wounds should be widely opened and free drainage established. If the opening of previous incisions is not sufficient or if the disease has spread beyond the area thus freely drained, multiple incisions should be made. Locally the Carrol-Dakin technique may be used. In Feeney's[9] report the advantage of using the Carrol technique with 1 per cent acetic acid instead of Dakin's solution is discussed. Supportive treatment consisting of fluids by hypodermoclysis or intravenously should be begun immediately; aid evacuation by enemata or low colonic irrigations; morphinization, gastric lavage, etc.

The efficacy of antitoxin treatment has not been established definitely. It is probable that it is of some value and certainly is worth giving so long as it is performed in

such a way that no untoward reaction occurs. The intradermal or eye test should always be done before an injection of any quantity is given. Injections should always be given slowly. The antitoxin is most rapidly effective if given by vein and when an infection is first discovered it is wise to give the first therapeutic injection by this route. Subsequent injections may be given intravenously or subcutaneously. Jennings[6] prefers to include antitoxin in either intravenous or subcutaneous fluids and has given as much as 1000 c.c. in twenty-four hours. Eckhoff[5] gives the antitoxin intravenously for its general effect and also injects from 25 to 50 c.c. locally at the site of infection. Probably the most effective antitoxin is the polyvalent, concentrated gas-gangrene antitoxin, including Perfringens, Vibrion Septique, Oedematiens (Novyi), Sordelli and Histolyticus Antitoxin.

In neither of the 2 cases reported here can the final outcome be attributed to the antitoxin used. In both cases it was felt that the antitoxin was of definite value to the patient involved but there was no specific or dramatic effect which could be positively ascribed to this therapy.

As is the case in practically every disease known, there are always to be found advocates of *x*-ray treatment. Gas-gangrene infection is no exception to the rule. Kelly[10] first reported the treatment of 6 cases of gas-gangrene infection with *x*-rays, and his work was followed up by Faust[11] who reports on 9 additional cases. All of Kelly's cases survived as well as 7 of those recorded by Faust. All of these cases were infections of the extremities following accidents. It is interesting to note that after the *x*-ray treatments were started it was not necessary to perform any amputations. Faust's other 2 cases died, one an infection in a limb, the other an infection of the trunk. In the latter case and *x*-ray therapy appeared to have no effect and the author believes that the small dosage used in limb affections is not sufficient to penetrate far enough in trunk lesions to be of value. His technique consists of a 5 inch spark gap, 5 ma., 40 cm. distance from target to skin, with 0.5 mm. aluminum as a filter. He recommends heavier voltage and filters to obtain the penetration necessary in trunk lesions. I have never seen this type of therapy used and so have no comments to make as to its efficacy. The reports available to date show a marked decrease in mortality where radiation therapy was used.

CONCLUSIONS

1. Two cases of gas-gangrene infection of wounds following appendectomy are reported.
2. The signs and manner of onset of this complication are briefly discussed.
3. Therapeutic measures, general, specific and *x*-rays, are outlined and briefly discussed.

For consultation and guidance and permission to render this report, I am indebted to Dr. F. W. Bancroft, Chief Surgeon of the Fifth Avenue Hospital.

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CYCLOPS

CASE REPORT*

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THIS case is reported because of its relative infrequency and the rarity of radiographic studies made in the literature.

FIG. 1.

J. G., aged thirty-nine years, para IV, registered at the prenatal clinic of the Greenpoint Hospital on January 21, 1931. Family and past personal history were irrelevant. Her menstrual periods began at the age of twelve years and were always regular. The last regular menstrual period occurred on June 15, 1930. The previous obstetrical history was normal. Subsequent prenatal visits revealed no abnormalities.

Labor began at 1:30 P.M. on March 24, 1931, and the patient was admitted at 5 A.M. Upon examination, it was found that the patient was in labor, the fetus in L.O.A. position, with the fetal heart in the left lower quadrant, regular, of good quality, and rate 128. Pelvic measurements were ample and her blood pressure was 118/80. The duration of the entire labor was nine hours, the second stage taking twenty-five minutes. The fetal heart disappeared during the first stage of labor. The cyclopic monster delivered spontaneously and was stillborn. The postpartum course of the mother was uneventful and all laboratory findings were normal.

Pathological Report (Dr. E. Koch). The specimen is a female fetus weighing 1500 grams. It measures 43 cm. from the vertex to the sole of the feet in extended position, the crown-breech measurement is 28 cm. and micro-

FIG. 2.

cephalic head measures 21 cm. in circumference. The ears are normally formed. This is a cyclops monstrosity with a single median eye, the optic fissure appears to have four definite segments, each representing a lid. There is no apparent canthus except in the lower angle. On external inspection the single eye appears normally formed. The lower angle of the optic fissure is 18 mm. above the mouth, and 4 mm. above the lower angle is the upper border of the optic fissure. Above the optic fissure is a median proboscis, the base of which is 8 mm. in diameter. The proboscis proper is 23 mm. in length, 12 mm. in its widest diameter, and 1 cm. from the base, with an encircling fissure. At its distal extremity, it presents a single pit like depression or cup which admits a probe

* Presented at a meeting of the Brooklyn Gynecological Society, held on November 1, 1935.

for a distance of 10 mm. There is no development of cartilage or bone apparent in association with the proboscis. There are no other external evidences of congenital anomalies. Necropsy was not performed so that the specimen might be preserved.

Radiographic Report (Dr. L. P. Van Winkle). A full term fetus showing no bone pathology in the left upper extremity, both lower extremities, spine and ribs. The right upper extremity shows abnormal development of the radius and ulna. The *skull* is of the microcephalic type. The bones of the vault are well developed. The soft tissues at the anterior aspect show the nose protruding at a level above the orbit. There is evidently only one large orbit located in the median line, radiographic evidence of cyclops.

SUMMARY

A para IV, having an uneventful prenatal course, spontaneously delivered a stillborn cyclopic monster. The important pathological findings were a single median eye above which was a median proboscis. Radiographic findings revealed a microcephalic skull containing one large orbit located in the median line.

THREE UNCOMMON TUMORS*

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THE following cases seem worthy of publication on account of their rarity and a hitherto undescribed giant cell type.

CASE 1. Peritoneal mesothelioma.

A man, aged forty-nine years, complained of gastric distress suggestive of peptic ulcer of nine months' duration. On *x*-ray examination a prepyloric filling defect with ragged outlines, the size of a cherry, was detected and the pylorus appeared to be transformed into a rigid, tapering tube. The diagnosis of carcinoma of the stomach was made and operation advised. At laparotomy, round, whitish, slightly prominent spots, varying in size from a millet grain to that of a lentil, were seen scattered throughout the entire peritoneum, but chiefly on the lesser omentum, which was studded with confluent spots. There was no free fluid in the abdominal cavity. At one point the lesser omentum was adherent to the anterior abdominal wall producing a kinking of the prepyloric region of the stomach but no tumor was palpated in the stomach. After severing the adhesion and removing a small piece from the lesser omentum for microscopic study the abdomen was closed in layers. The patient was discharged from the clinic on the eleventh postoperative day.

Microscopic Examination. The surface was covered invariably by a continuous single layer of regular, cuboidal cells. Except for insignificant breaks of continuity, this cell layer exactly followed all prominences and recesses of the surface (Fig. 1). In the deeper layers, several branched formations are seen resembling buds of capillaries, however, containing no blood. (Fig. 2.) From the surface swarms of cells, identical with the covering cells invaded the deeper layers, always remaining single and isolated without any tendency to form epithelial structures, as glands or cells nests, according to the term, "invasion à cellules isolées," for this type of infiltration. The tumor cells were rather large and somewhat oval. Their sides, looking towards the surface, were vaulted, whereas the others in contact with the neighboring cells were flattened. The nuclei were relatively small and surrounded by a rather broad zone of cytoplasm. (Fig. 3.) In the remainder, the nuclei were round or slightly oval, clear structured, with a distinct nuclear membrane and nucleolus. The cells showed almost no polymorphism and though there were some variation in their size and even numerous considerably larger cells than the average containing two to three nuclei, the cell type was fairly uniform and resembled conspicuously the swollen endothelium of lymphatic sinuses. There were very few typical mitoses but several nuclear forms suggesting amitosis, were observed. In the fibrous and fatty tissue beneath the lining layer were groups of lymphocytes and scattered plasma cells, no other signs of inflammation, however, being present. The endothelial lining of the blood vessels and lymphatics showed no proliferation.

Among special staining methods, Weigert's elastic tissue stain demonstrated the presence of a plexus of slender elastic fibers beneath the lining layer. With methyl green-pyronine, the cytoplasm of the tumor cells stained distinctly red, though not as brightly as do plasma cells. The Giemsa stain failed to demonstrate azurophilic granulation.

Primary tumors of the peritoneum, considering only those arising from the lining cell layer and excluding all others originating from the subserous or retroperitoneal tissue, as fibromas, myxomas, sarcomas, etc., are quite rare. The overwhelming majority of the cases described in the literature are papillary, tubular or alveolar tumors, i.e. producing epithelial structures, (Rokitansky,[1] Borst,[1] etc.). Waldeyer[1] reported a case of angiosarcoma. In a few cases the tumor cells showed an endothelial behavior, they covered the surface and their cytologic type, too, was endothelial

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(Kaufman,[2] Kux,[3] Herzog,[4]) in the depth, however, they invariably tended to form tubular or alveolar structures. We were unable to find a second case in which the covering cells so strictly retained their endothelial character as they did in our case.

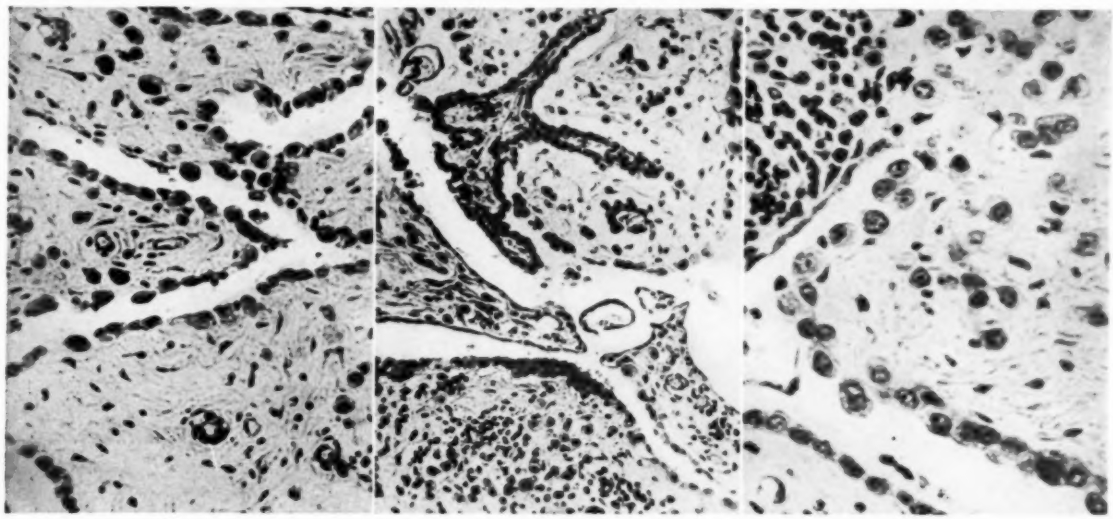

Fig. 1. Fig. 2. Fig. 3.

Fig. 1. The surface is covered by a single layer of endothelium like cells. × 200.
Fig. 2. A branched formation, resembling young capillary. × 120.
Fig. 3. Morphology of lining cells, high power magnification.

As to the rational denomination of tumors of this type, opinions differ. Those who consider the lining of the pleural and peritoneal cavities to be epithelial, according to the celoma-theory of Hertwig, will speak of carcinoma. Adherents of the endothelial origin of the lining cells, following the theory of His, will consider these tumors to be endotheliomas. According to more recent embryologic researches the pleuroperitoneal cavity results from a cleft formation in the mesoderm and the lining cell layer is a product of secondary differentiation. These cells are enabled to differentiate into epithelium as well as connective tissue. Therefore Marchand, and after him Kux, consider a most appropriate, the unbiased term mesothelioma or malignant lining cell tumor, "maligne Deckzellengeschwulst," reserving the term "endothelioma" only for those tumors actually arising from the endothelium of the blood vessels or lymphatics. Both denominations have been accepted in the medical literature, therefore we feel justified to call our case a peritoneal mesothelioma.

A few months later the patient reappeared at the clinic with obvious signs of progressive cachexia. His further fate is unknown to us.

Case II. Osteo-osteoidsarcoma of the breast.

A woman, aged sixty-four years, complained of a painless lump in her right breast of six months duration which grew slowly but continuously until it reached its present size of a newborn baby's head. A month ago a sore developed on it. For two days prior to admission her breast was red, swollen and painful. The patient was well developed and somewhat obese with huge, pendulous, fatty breasts, each larger than a man's head, the right being much more voluminous. In its lower outer quadrant was a firm lump about the size of a child's head. Just below the nipple there was an ulcer, 3 cm. in diameter, of cartilaginous consistency, with a fungating floor, raised borders surrounded by a broad zone of inflamed, edematous skin and discharging foul smelling, purulent material. The subcutaneous veins for a great distance were bordered with red streaks. No lymph glands were felt in the axilla. The temperature was 39.5°C.

After treatment with wet acriflavine dressings the inflammation subsided within four days and the temperature dropped to normal. On the tenth day the patient was operated for

sarcoma of the breast. The breast was amputated including the fat tissue and excision of the lymph glands of the axilla, but the pectoralis muscles were not removed. The skin was closed with interrupted silk sutures and a rubber drain inserted into the wound. Recovery was uneventful and the patient left the clinic on the thirteenth postoperative day, having a small granulating wound the size of a bean, at the site of the drain tube.

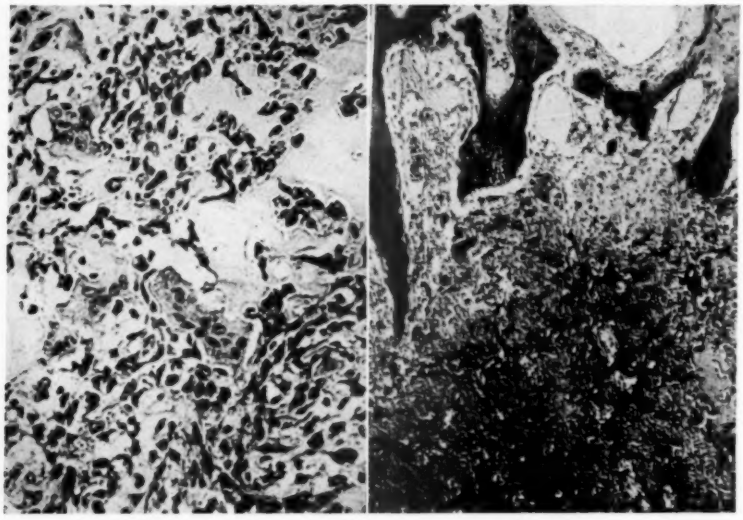

Fig. 4. Fig. 5.

Fig. 4. Osteoid sarcoma with giant cells. × 120.
Fig. 5. Bony trabecula in the tumor tissue. × 50.

When attempting to halve the operative specimen the knife met calcareous resistance necessitating sawing. The cut surface showed a grayish white, roughly lobulated tumor of firm consistency occupying the greatest part of the breast. In its substance there were many reddish areas, ranging from the size of a hazelnut to that of a walnut, resembling cancellous bone. In the softer parts of the tumor were crumbling, necrotic cavities, the ulcer communicating with one of the large cavities. The entire tumor was close to the skin being separated from the pectoralis fascia by a two fingers thick layer of normal fat tissue. The axillary lymph nodes showed no involvement.

Many blocks were cut from different parts of the tumor for microscopic study. The calcareous parts were partly decalcified with sulphosalicylic acid and partly impregnated with my own method.

The microscopic picture was intensely interesting. In places it showed a rather fibroplastic spindle cell sarcoma with many areas of necrosis. The polymorphism of the cells was moderate, mitoses were scanty. The connective tissue strands coalesced frequently to form broad, clumsy trabecula, arranged into a rough network. Their substance stained red with the van Gieson stain. In their interior but very few cells were seen. Their surface, however, was lined with spindle shaped or oval cells, as a rule forming a single layer, but islets of these cells occurred also. In the cellular areas between the trabecula there were many polynuclear giant cells of the bone marrow type. (Fig. 4.) In several places the strands of thick connective tissue blended gradually into regular bony trabecula, corresponding to normal bone. The cellular sarcomatous tissue ended rather abruptly at the confines of the bony transformation with only a fibrous bone marrow tissue between the trabecula. No hematopoietic bone marrow was observed throughout the sections. In other places the sarcomatous part was separated sharply from normal looking bone tissue by a capsule like layer of compressed connective tissue. (Fig. 5.) Preparations stained according to my method* showed the bony trabecula to be completely calcified. Many calcareous granules were scattered throughout the osteoid trabecula, too, in places forming bulky masses, (Fig. 6 and 7), chiefly near the juncture of the bony transformation. The lymph glands examined showed signs of a low grade inflammation. According to the microscopic finding the tumor is to be classified as an osteo-osteoidsarcoma.

Sarcoma of the breast is a rare condition; according to statistics its frequency

* Gömöri: *Am. Jour. Path.*, 9: 253, 1933.

amounts to a few per cent of carcinomas and those containing bone tissue are so exceptional that textbooks of pathologic anatomy, e.g. that of Aschoff, often fail to mention their existence. Several instances of smaller or greater islands of cartilage or bone in pure mesodermal or in mixed, or ecto-mesodermal, tumors of the breast have been described, but in almost all of the cases the bone tissue found was abortive, rudimentary or at least atypical. Only 6 cases of well developed bone tissue, as a characteristic element of the tumor, could be found in the literature available to us; one case each of Arnold,[5] Heurteaux,[6] Hueter-Karrenstein,[7] Kaufmann,[8] 2 cases of Stilling.[9] In Gross'[10] case of carcinoma simplex there were well developed bony trabecula embedded in the stroma. Osteoid sarcoma of the breast, of both the calcifying and the non-calcifying varieties, is also extremely rare (Stilling,[9] Schultz-Brauns,[11] Edelmann[12]).

As to the origin of similar tumors not much is known; they probably take their starting point from aberrated skeletal germs.

Fig. 6.

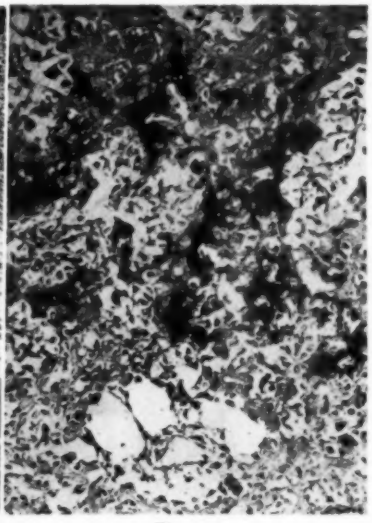

Fig. 7.

Fig. 6. Low power view of the tumor. In the connective tissue separating the lobules of the tumor are lactiferous ducts. In the central parts of the osteoid trabecula are seen black staining lime salts (Gömöri silver impregnation, hemalum-eosin stain). × 50.

Fig. 7. Calcareous deposits in the osteoid trabecula (black). Staining as in Fig. 6. × 120.

Case III. Xanthoma of tendon sheath.

A woman, aged thirty years, complained of a painless lump on the dorsum of the root of the second toe of the left foot. It grew slowly for several years until it attained the size of a small walnut. On admission, there was a freely movable, oval swelling of fleshy consistency not adherent to the skin. All other findings were normal. The tumor was easily enucleated except for rather firm adhesions binding it to the extensor tendon. The removed specimen had the shape and size of an average prune with a lobulated surface and a delicate, unbroken capsule. The cut surface had a tan color. Many blocks were cut for microscopic study.

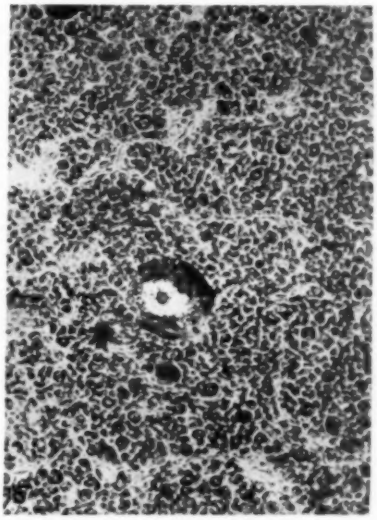

Fig. 8. Cellular, tumor like tissue with giant cells. × 100.

The microscopic picture was not quite uniform. At the center of the specimen, a highly cellular tissue, lobulated by fibrous stroma, was seen. The cells were round or polyhedral and varied in size. The cytoplasm of most of the cells was packed with yellowish brown granules which formed a broad, dense ring around the vesicular nucleus. As they gave the prussian blue reaction, they evidently consisted of hemosiderin. Mitoses were very few. Amidst this groundwork of smaller and medium sized cells were many giant cells, the biggest being about 140 to 160 microns in diameter, (Fig. 8) with numerous small round or oval, rather deeply staining nuclei, grouped either around the periphery or forming a garland nearer the center of the cell. In the latter case the cytoplasm peripherally from the zone of the nuclei had a foamy structure, whereas its central part was homogenous. Giant cells of this second type, exactly corresponding to that described by Touton[13] were much less numerous than those of the first type, resembling foreign body giant cells. The latter were often packed with hemosiderin granules. In a number of these giant cells of the foreign body type were queer formations, never seen or heard of by us before. In a clear, foamy structured area, surrounded by the garland of nuclei a distinctly outlined, many-rayed star was seen, its center forming a disk showing concentric layers, from which as many as 20 to 40 processes were radiating. In some of the stars the central disk was bulky, the processes were short and clumsy (Figs. 9 and 10); in others long, spider arm like, tender processes radiated from a central granule and almost reached the periphery of the foamy area (Fig. 11). Several giant cells contained two clear areas, each with a star. The stars stained dull red with eosin, orange yellow with van Gieson's stain, the center being darker than the rays. They contained no lime salts, did not stain black with iodine and sulphuric acid nor showed any metachromasia with either methyl or cresyl violet. They were stained very distinctly with iron hematoxylin. At the periphery of the tumor the microscopic picture differed. Nests of clear, foamy cells with a small, deeply staining nucleus were seen in a network of broad connective tissue strands.

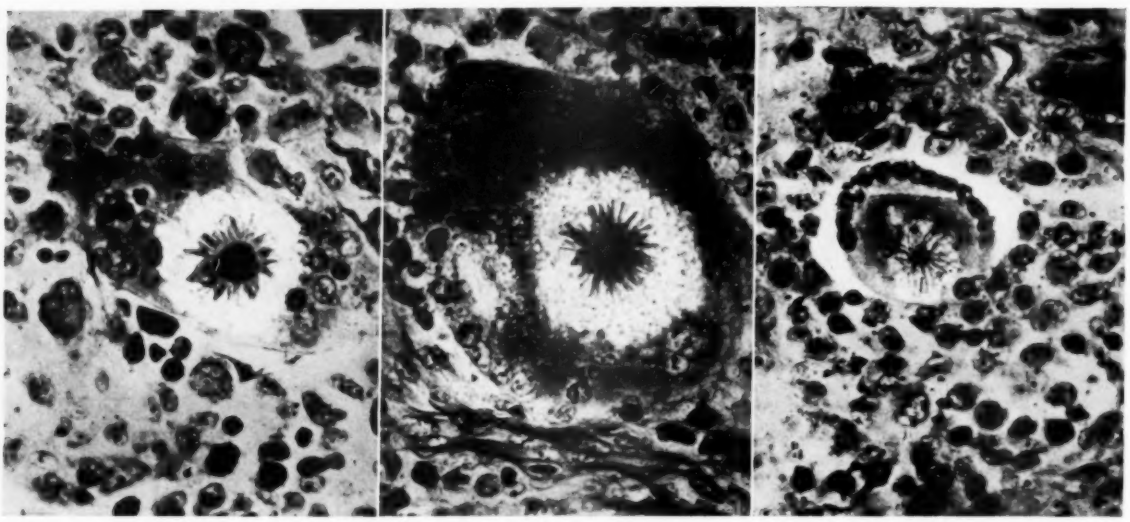

Fig. 9. Fig. 10. Fig. 11.

Figs. 9, 10 and 11. A giant cell with star. High power magnification, iron hematoxylin stain. × 500.

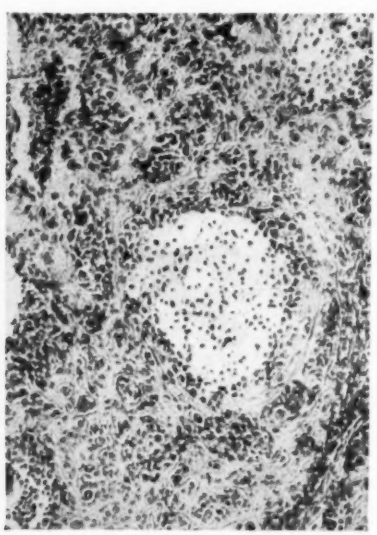

Fig. 12. Nests of xanthoma cells. × 100.

(Fig. 12.) In some of these nests only the lining cell layers remained intact, the central cells having undergone necrosis and liquefaction. Many connective tissue spaces were packed with hemosiderin laden round cells and mostly with foamy cells. In preparations stained with Sudan III the foamy cells were seen filled with orange red fat granules. However, numerous fat granules were present also in cells showing no foamy structure and in many cells of the connective tissue bundles. The clear area surrounding the stars, too, was made up of fat droplets. The stars themselves did not stain with Sudan. From the center of the tumor radiating towards the periphery, the microscopic picture showed a gradual transition. The entire nodule was encapsulated and the pathologic tissue did not extend beyond the capsule. No inflammatory signs were observed.

We are unable to form a decisive opinion concerning the stars. They were by no means accidental artefacts or inclusion bodies as they were too numerous and of a conspicuously uniform morphology. There was no question about their being parasites. Nothing was left but to consider them as products of degeneration, probably in connection with disintegration of some fatty material. As to their chemical composition staining did not yield any information.

Following the operation the patient was well for six weeks. When there was a recurrence, the size of a walnut. At second operation a similar, lobulated tumor was found, firmly adherent to the extensor tendon. Grape like prolongations of the tumor pervaded the interosseous muscles as far as the sole of the foot. It was impossible to dissect them individually, therefore the tumor was removed "en bloc" together with a segment of the tendon, measuring 4 cm., and a great part of the interosseous muscles. After healing of the wound the patient received a heavy series of *x*-ray irradiations. Four months after the second operation the patient was well and free from recurrence.

The microscopic picture of the growth removed at the second operation was identical with that of the first tumor. The relationship of the tumor to the tendon was interesting. At places it was separated from the surface of the tendon by a layer of connective tissue, at other places, however, it invaded the more superficial layers. In the tendon many groups of hemosiderin laden cells were seen at considerable distance from the tumor.

We do not wish at this occasion to deal in detail with the extensive literature of the xanthoma problem. It is sufficient to state that there are pathologic formations of identical microscopic structure, (foamy cells, giant cells of the Touton[13] type, hemosiderosis) which may be (1) symptoms of a faulty metabolism, as hypercholesterolemia, manifesting itself in multiple cholesterol deposits or xanthelasmata; (2) multiple, tumor like nodules, chiefly around joints and tendons, without disturbance of metabolism; (3) single tumor like formations on the tendons of the hand or foot, without disturbance of metabolism. Pathologic conditions belonging in the first group were never the object of discussions, their non-neoplastic nature being universally accepted. On the contrary, formations belonging in the second and third groups are a matter of dispute for decades and the question is not settled as yet. Some students of this problem are explicit adherents of the neoplastic theory, as malignant tumors of this kind showing infiltrative growth and rapid recurrence after removal have been recorded by Chassaignac,[14] Billroth,[14] Coenen,[15] Fritsch,[16] Rosenthal[17] and others. Metastases, however, were never observed. Others as von Albertini[18] and Nachtnebel,[19] while insisting on their neoplastic nature, emphasize their benign character. Still others reject the neoplastic theory; Wustman[20] speaks of cholesterol tophi, Fleissig[21] and Pommersheim[22] of traumatic tendon sheath granuloma, possibly on a constitutional basis. It should be mentioned that the theory of traumatic granuloma is not supported by the fact that xanthomas of the foot are a most unusual condition in comparison with the great frequency of other diseases of traumatic origin of the lower extremity. Herxheimer uses the rather vague term "granuloma sarcoides xanthomatosum." According to Wegelin[23] the process is an inflammatory-resorptive one, caused by necroses. Pick and Pinkus[24] reject the assumption that xanthoma is a distinct pathologic entity; in their opinion,

under certain circumstances any normal or pathologic tissue is liable to undergo xanthomatous transformation. Therefore the use of the attribute "xanthomatosum" is justifiable together with the name of the tumor concerned. Lack of space precludes discussion of other theories.

We wish to add the following considerations: The outlines of the concept of neoplasm are not absolutely sharp as yet. There are many pathologic conditions at the border zone of the realm of neoplastic diseases the exact nature of which is debated, because conclusive, unequivocal criteria are lacking; for example, it is sufficient to mention Hodgkin's disease, the leukemias, multiple osteochondromata, neurofibromatosis, ganglions, so-called giant cell sarcomas of bones, tumor like embryonal malformations. Among these the resemblance between giant celled bone tumors and xanthomas is striking. The microscopic picture of both conditions is similar: tumor like tissue containing giant cells; hemosiderosis; foamy cells containing cholesterol. Though in bone tumors the latter are not as common as in xanthomas, they are by no means exceptional and at our clinic there was also a giant celled bone tumor in which foamy xanthoma cells abounded. Both conditions have two clinical forms: a localized and a generalized one, the microscopic picture, however, of both forms being identical. The localized tumor like forms are giant celled bone sarcoma and solitary xanthoma, respectively; the generalized forms are dependent on a disturbance of metabolism, as Recklinghausen's cystic disease of the bone with disturbance of calcium and phosphorus metabolism; xanthelasmata or xanthomatosis with disturbance of cholesterol metabolism. Both diseases, chiefly in their localized forms, show a preference for the younger age. The localized forms of both diseases resemble true neoplasms, recurrence after removal is frequent, but no metastases are observed with either of them. Of course, the pathologic nature of giant celled bone "sarcomas," too, is a moot point; in his lecture held at the Hungarian Medical Week in Budapest in 1933 Puhr defined them as tumor-imitating neo-formations of the reticuloendothelial system. Going into the details of the pathology of tumor like giant celled conditions would lead too far, it is enough to state that, on the basis of our present day knowledge they find their most acceptable, though not quite satisfactory place under the heading of benign tumors. As to their surgical treatment it should be borne in mind that tendon sheath xanthomas, though they may seem malignant on histologic examination and though they will occasionally recur after removal, they never will give rise to metastases, therefore too great radicalism in their removal is not justifiable.

SUMMARY

Three unusual cases, a peritoneal mesothelioma, osteo-osteoidsarcoma of breast and xanthoma of tendon sheath are reported with detailed microscopic description and discussion of each.

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NEW INSTRUMENTS

NEW SKIN THERMOMETER

FOR DIAGNOSIS OF PERIPHERAL VASCULAR DISEASE*

WILLIAM S. COLLENS, M.D. AND NATHAN D. WILENSKY, M.D.

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RECENT developments in the knowledge of peripheral vascular disease have been greatly accelerated by the measurement of the skin temperature. Gibbon and Landis[1] have applied a long known principle to clinical studies of peripheral vascular disease in an attempt to differentiate between peripheral vascular spasm and organic occlusion. They record the temperature of the skin on the dorsum of the distal phalanges after the extremity has been exposed to room temperature for approximately one-half hour for purposes of equalization. The upper extremities are then immersed for about twenty minutes in water at 109° to 113°F. In the normal response there is an elevation of the skin temperature of the lower extremities of approximately 20°F. at the end of one-half hour. When organic disease of the peripheral arteries exists, there is marked interference with this vasodilator reflex response.

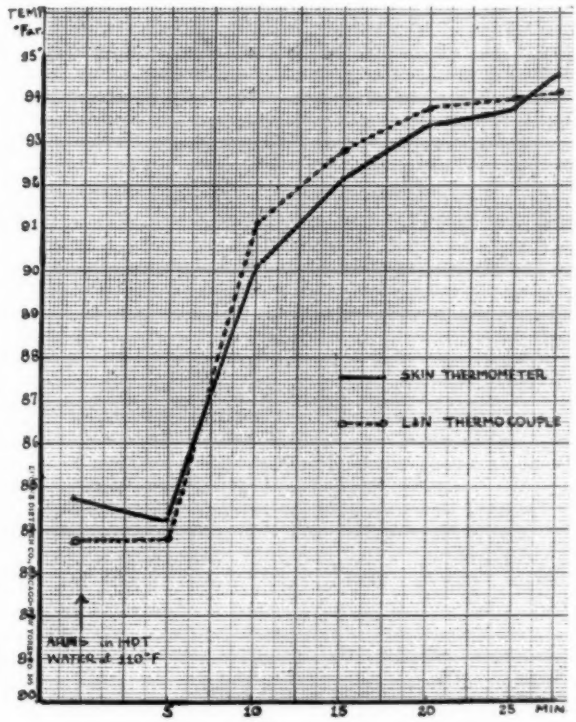

FIG. 1.

FIG. 2.

This test has assumed great clinical importance in differential diagnosis and an accurate interpretation of peripheral vascular disease can hardly be performed without it. The chief difficulty, however, exists in the fact that, up to the present time,

* From the Metabolic Clinic of the Israel Zion Hospital.

recording of skin temperatures could only be performed by means of complicated and expensive thermocouple equipment.

We have devised a simple thermometer which is extremely accurate and very inexpensive and with which skin temperature readings can easily be taken in the performance of the Landis test. The device consists of a mercury thermometer, similar to the ordinary clinical thermometer but with a finer bore to permit a rapid rise of the mercury column. The bulb of the thermometer is encased in a Bakelite cap but one surface of the bulb is exposed which is used for contact with the skin. The glass column of the thermometer is 8 inches long and graduated in 0.2° increments from 75° to 105°F.

The accuracy of this thermometer was established in comparative studies with a Leeds and Northrop thermocouple in the Vascular Clinic of Dr. Irving S. Wright at the New York Post-Graduate Hospital. The observations were made on a normal individual in a room whose temperature was 81.5°F. The arms of the subject were immersed in a bath at 110°F. The skin temperature readings were taken on the dorsum of the right foot just proximal to the base of the third toe. The comparative results are seen in the accompanying graph. (Fig. 1.)

It will be observed that the skin thermometer was capable of recording the same degree rise in temperature as the thermocouple. The total deviation was 0.1° in a total rise of 10.4°F.

The method of application of the thermometer can be seen in the photograph. (Fig. 2.)

CONCLUSION

A new type of skin thermometer is presented which is as accurate as the electrical recording device and is so reasonable in price as to make it possible for the general practitioner to introduce this procedure in the differential diagnosis of peripheral vascular disease in his private practice.

We are indebted to Dr. Wright for assistance in making the comparative studies.

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MODIFIED OCHSNER TROCAR

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WASHINGTON, D. C.

THE Ochsner trocar that is usually employed to evacuate the gall bladder is a very useful instrument and has a variety of uses other than the one for which it was designed.

On a number of occasions we have been annoyed with the tendency of the plunger to fall in the canula and thereby occlude its lateral opening, attention being called to this occurrence by the sudden arrest of the flowing stream of aspirated fluid. By lifting the plunger, the aspiration will be renewed. To avoid this unnecessary interruption we have designed a trocar in which the plunger can be held in position by a turn and released as easily. The illustration permits this explanation. By means of the thread (*a*) with one or two turns the trocar can be kept in its correct position and cannot possibly prevent the escape of fluid through the canula. The graduation of the canula (*b*) serves the purpose of giving in centimeters the penetration of the instrument. Not infrequently a trocar has been withdrawn unintentionally because the location of the end was not known. The finger hold (*c*) merely allows better manipulation of the instrument.

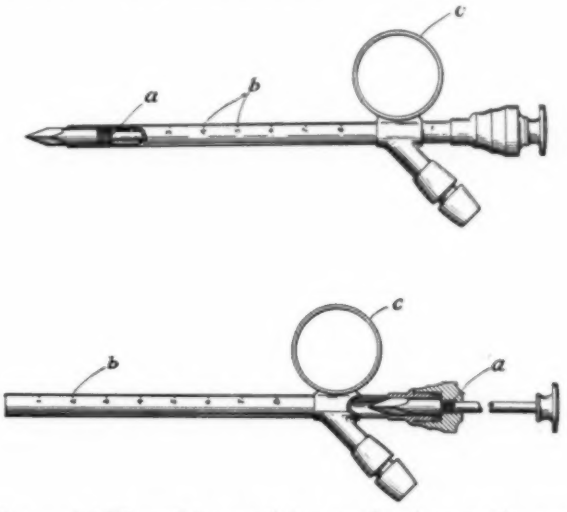

FIG. 1. (*a*) Thread for retaining trochar in a stationary position within the canula. (*b*) Graduations. (*c*) Finger hold for better manipulation.

We have been using this instrument in our local hospitals for three years or more and it has largely replaced the former type because of its proven advantages.

[From Fernelius' Universa Medicina, Geneva, 1679.]

BOOKSHELF BROWSING

COSMAS AND DAMIAN, PATRON SAINTS OF SURGERY*

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TIME brings with it not only changes in manners and customs but also in the objects of our veneration. Thus it is, that saints who, for centuries, were patrons of the profession of surgery are now unknown, even by name, to most of its votaries, and in the current histories of the craft are granted but a grudging footnote, if mentioned at all. It is the purpose of this paper to briefly recall the legends dealing with the lives and miracles of Saints Cosmas and Damian, and to trace the rise of their cult as patron saints of surgery.

Cosmas and Damian, according to the more popular of these legends, were born in Arabia, in the third century A. D., the eldest of five sons of Christian parents, and passed most of their lives in Aegea, on the Gulf of Issus in Cilicia in Asia Minor. Their mother, the devout Theodota, reared them in the faith, imbued them with the Christian virtues and taught them the Holy Script. They became physicians and, in healing the sick, combined physic with prayer. Because of their piety and virtue, their efforts were blessed and they were credited with many marvelous cures. Their following was large and included not only suffering humanity, but domestic animals as well. In keeping with the saintliness of their characters, they vowed to accept no fees for their services, which won for them their appellation, *anagyres*, meaning without silver.

So firmly were the brothers committed to the code of gratuitous practice that it led to an estrangement between them. A woman named Palladia, suffering from an illness which baffled all other physicians, sought their aid. She was promptly healed, whereupon she prevailed upon Damian to accept three eggs in grateful recompense. Cosmas was so perturbed at his brother's apparent fall from grace, that he declared he would not be buried with him. However, the Lord appeared to Cosmas in his dreams that night, and explained that Palladia had vowed to Him to bestow the eggs on whomsoever effected the cure and it was in order to keep her from breaking a holy vow, rather than because of his desire for the reward, that Damian had violated his own oath and accepted the eggs. Cosmas was appeased by the divine intercession and retracted his decision to be separated from his brother after death, but failed to inform his friends of this change of heart. When, in

* Read before the Society of Medical History of Chicago, March 29, 1935.

the course of time, the brothers were martyred, preparations were made to inter them separately. A camel which had once been cut in half by the devil and made whole again by Cosmas, appeared at the burial and pleaded in perfectly grammatical Latin, "Nolite eos separare," (Let them not be separated) and told of the change in Cosmas' feelings toward his brother. The assembled folk acted upon the advice of the camel, and the brothers were buried together, a detail which greatly simplified the subsequent search for relics of the twin saints.

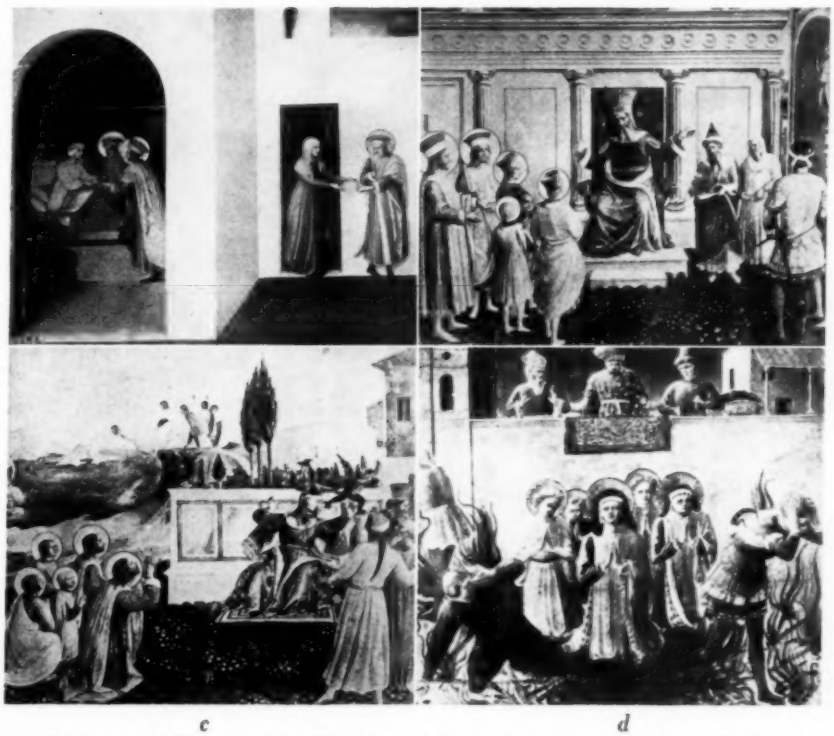


FIG. 1. Scenes from the lives of Cosmas and Damian. Paintings from the School of Fra Angelico. *a*. Damian accepts three eggs from Palladia. *b*. Before the judge. *c*. Rescued by an angel from drowning. *d*. On the fire which refuses to burn them.

The fame of the Christian physicians and the tales of their miraculous cures reached the ears of Lysias, governor of Aegea. They were haled before the tribunal, where they proclaimed their faith and refused to make obeisance to the pagan gods. They were sentenced to be flogged but fortified by their supreme devotion, they rejoiced in their tortures and asked for greater punishment. Their death was then ordered. Bound in chains, they were cast into the sea but an angel released the bonds and drew them to shore. They were placed on a burning pyre but the flames refused to harm them, although several of the onlookers were burned. They were then ordered stretched on crosses and pelted with stones but the missiles refused to find their mark and recoiled to strike the throwers. Even arrows shot at their defenseless bodies, like boomerangs, turned to wound the archers. Finally, in desperation, Lysias demanded that they be beheaded, and, as was usually the case, this simple expedient proved effective. These events have all been portrayed in paintings (Figs. 1 and 2), in many of which, the three younger brothers of the saints are also shown, and in some, they share the martyrdom.

The synaxarion of the Greek church recognizes three pairs of saints named Cosmas and Damian, with a distinct legend and their own calendar day for each

pair. According to the Asiatic tradition, which is the oldest of the three, the saints died natural deaths and were buried in Pelusion, in Egypt. The Roman and Arabian tales crown the lives of the saints with the martyr's death, the latter version being the only one recognized by the Roman church. It is this Arabian legend which is sketched, and which forms the basis for the western cult of the twin saints. According to it, after their martyrdom, Cosmas and Damian were buried in Cyrus, in northern Syria. The earliest miracles associated with the names of Cosmas and Damian occurred at this tomb, and their cult spread, by way of Byzantium, to Europe.

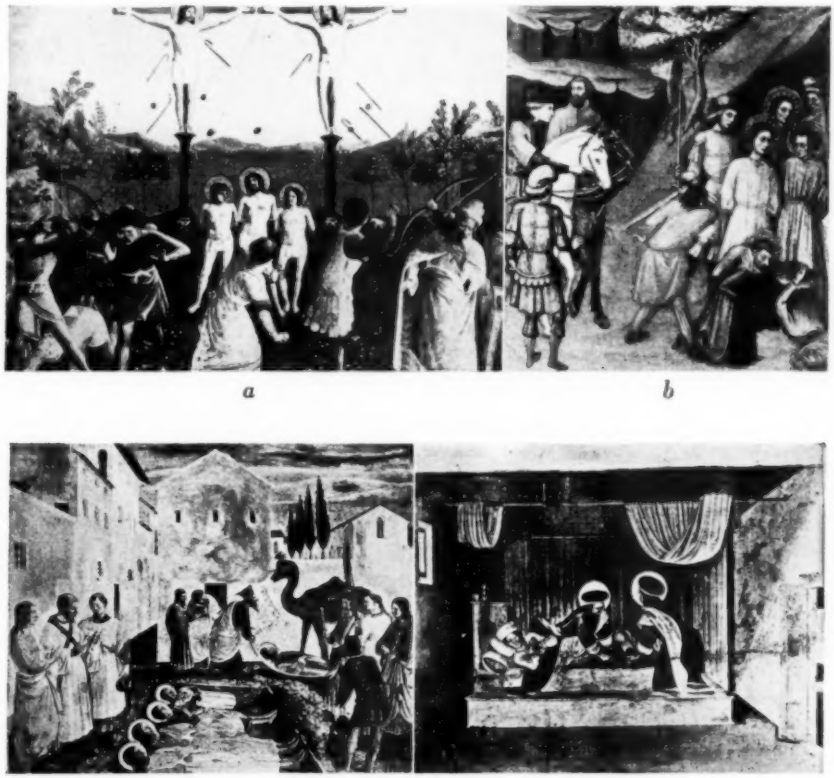
Hagiologists are in accord that the legend of Saints Cosmas and Damian is a Christian adaptation of an earlier pagan cult, and that the martyred physicians are entirely mythological figures, created for this purpose. This, of course, in no way diminishes their claim upon our interest, nor minimizes the part they played as the patron saints of the healing professions. Harris[11] has shown that Saints Cosmas and Damian represent a Christian version of the ubiquitous cult of the heavenly twins, as embodied in the Greek Castor and Pollux, or the Asvin twins of the Vedic mythology. The latter, incidentally, also practiced supernatural healing, even among the gods themselves. Deubner[5] believes the story to be borrowed from the legends of Zenobius and Zenobia. The multiplicity of the Cosmas and Damian legends is ex-explained by the tendency of chauvinistic biographers to honor their own countries by placing within their boundaries the scene of activity as well as the burial place of the famous saints. It is likely that the tomb of Cosmas and Damian was officially placed in Cyrus because that city was already the seat of a famous Aesculapian temple.

With the impetus derived from the fame of its predecessor, the cult of Cosmas and Damian developed very early, and many posthumous miracles were recorded. For example, a farmer is said to have fallen asleep beneath a tree with his mouth open and an adder crawled down his throat and into his stomach. All efforts to dislodge the intruder were unavailing until finally, he prayed for relief at the tomb of the martyred physicians. He fell asleep and while he slept, the saints exorcised the serpent. By repeated miracles their shrine became so famous, that within two centuries after their death the Emperor Justinian sought healing there. Afflicted with a grievious ailment that had resisted all previous treatment, he prayed there and was cured promptly. In grateful acknowledgement he built a temple to Cosmas and Damian and caused the city of Cyrus to be fortified and beautified, and equipped with a water system. Later he had the remains of the saints brought to Constantinople, and built a basilica to them there. The cult spread rapidly through the East and appeared in Rome as early as the fifth century. Cosmas and Damian are included in the Canon of the Roman mass, having been introduced from the East before the rites of canonization were developed. They are the only oriental saints to be so honored.

The first basilica in Rome to Saints Cosmas and Damian was built by Pope Felix IV (526–530), on the Sacred Way, near the Forum. This church was built upon the ruins of, and with the materials from two pagan temples, and here the relics of the saints are said to have been brought. The church was rebuilt on the same site in 1630 by Pope Urban VIII and is still standing. In the present edifice are preserved some mosaics from the original Cosmas and Damian church. These sixth century mosaics, among the oldest in Rome, reveal an early Byzantine influence and are highly reminiscent of Pompeian art.

In this church, the most famous of the posthumous miracles of Saints Cosmas and Damian were performed. A man with a cancerous leg sought assistance from the saints and after praying at their shrine, fell asleep in the sanctuary. The saints

appeared in his dream and amputated the diseased member. To replace the lost limb the saints borrowed a leg from a dead Moor inspecting the grafted leg, while on the floor may be seen the diseased one which has been removed. In the distance a group of



a *b* *c* *d*

Fig. 2. Paintings of the lives and miracles of Saints Cosmas and Damian. *a*. Recoil of the missiles. (Fra Angelico.) *b*. Beheading. (Spinello Aretino.) *c*. Burial. (School of Fra Angelico.) *d*. Black leg grafted onto white patient. (Fra Angelico.)

who had just been buried in the churchyard of San Pietro in Vincole, and grafted it in place of the diseased one. When the patient awoke and found himself whole, he told of the miraculous cure. His incredulous friends hastened to the coffin of the Moor, and found, indeed, the limb to be missing.

This legend has provided the subject for many paintings, and is of particular interest in revealing the persistence of temple incubation sleep as a means of healing extending late into the Christian era. In the reproduction in Figure 3, the divine surgeons are seen cleaning their instruments after the completion of the operation. The patient appears to be sleeping quietly, the expression on his face suggesting narcosis induced by some sleep producing drug. The awe-stricken spectators are people stand in amazement about the open casket of the Moor, and close inspection will reveal that the corpse really has but one leg.

The original of this picture is a miniature in an old choir book now in the possession of the Antiquarians' Society of London, and has been attributed to Mantegna. This attribution has been doubted and it is considered more likely to be the work of a master of northern Italy, perhaps Ferrara, of the latter part of the fifteenth century. The version of the same operation in Fig. 2*d* was painted by Fra Angelico and now hangs in the Ospizio of San Marco in Florence. Another by Pesellino (1422–1457) is in the Louvre. From the surgical point of view neither of these is as interesting as the first one described. The large painting

reproduced in Figure 4 is unique in showing the operation anteriorally rather than in profile. The leg has just been amputated,

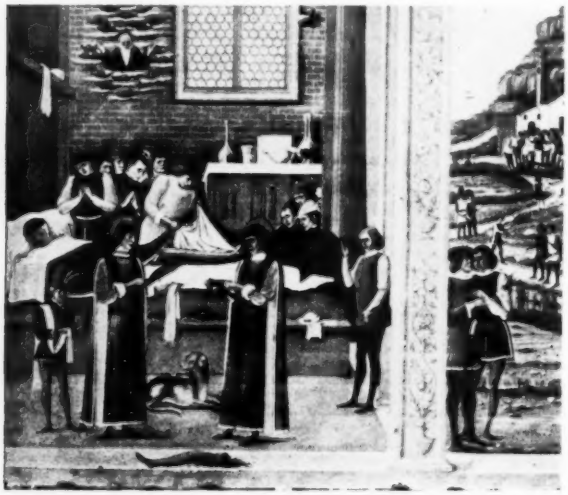


Fig. 3. Saints Cosmas and Damian amputate leg and replace it with limb of dead Moor.

the tourniquet is still in place, and the surgeon on the right is allowing the soft parts, which he has been retracting upwards, to fall back and cover the bone. The other saint has the black leg in his hands and is about to affix it to the stump. The diseased leg, and the saw that was used in removing it are shown in the foreground. The operation is apparently being performed in a hospital. In the background are seen ambulant patients receiving treatment and a physician inspecting a urine flask. To the left, a recumbent patient is being tended by a nurse. This picture, attributed to the elder Francken (1542–1616) now hangs in the museum at Antwerp.

Besides the basilica on the Forum, other churches in Rome were dedicated to Saints Cosmas and Damian. The cult spread rapidly through the rest of Italy and to other countries of Europe, so that as early as the sixth century, churches dedicated to the saints appeared in France and Belgium, and by the seventh century they were known in Germany, Spain and even England.

Because of their medical activities during life and the cures accredited to them after their death, Saints Cosmas and Damian soon became the patron saints of the physicians and also of the allied crafts,

Fig. 4. Miracle of black leg grafted onto white patient. (Francken.)

including the barbers, surgeons, apothecaries and midwives. In the Via de'Barbieri, in Rome, a church to Cosmas and Damian was the place of meeting and worship of the Barbers' Guild for many years, and even today the saints are the patrons of the barbers in Italy. In later medieval times, with the organization of the crafts into companies and guilds, Cosmas and Damian played a particularly important part as the tutelary saints of the barbers and surgeons.

In addition to their role as the patron saints of medicine and surgery, Cosmas and Damian were the patron saints of the

Medici family of Florence, and Cosimo de Medici is said to have been named for Cosmas. Another version is that Cosimo chose the saints for himself and his family because of the similarity of their names. In either event, he commissioned many works of art portraying the lives and miracles of Cosmas and Damian. This is one reason for the popularity of the saints in the paintings of the Renaissance masters, particularly those of the Florentine school of the fifteenth century. Fra Angelico and his pupils executed a series of nine panels based on the lives and legends of the saints, which were intended for the high altar of San Marco in Florence; several of these scenes are reproduced in Figures 1 and 2. He also included the saints in his Crucifixion. Fra Lippo Lippi has represented them in the group around St. John and they appear in the works of Titian, Tintoretto, Roger van der Weyden and others. A statue of each of the saintly brothers stands on either side of the door into the famous Medici chapel in Florence.

In paintings, the saints are usually depicted as men of intelligent and studious aspect, often shown with books or instruments of their profession. They frequently hold mortar and pestle or ointment box and spatula and occasionally, surgical instruments. The palm branch, symbol of the martyr's death, is also shown at times. In many pictures, one of the brothers holds a urine flask, indicating that he is a physician, the other is represented as a surgeon, usually with gallipot and spatula. In such instances the physician is usually clad in elegant ecclesiastical vestments, while the surgeon, more plainly attired, is in lay clothes. Many prints, particularly of the early German school, make a point of showing the medical men in long, flowing robes, while the surgeons wear short kilts, emphasizing the difference in social position of the practitioners of the two branches of the healing arts at that period. Where no distinction is made between the specialties of the two healers, they are both dressed in long robes, often trimmed with ermine. Their heads are usually covered. In many paintings the portraits of actual physicians have been used in representations of their saintly patrons.

In keeping with their vocation as healers, Cosmas and Damian had considerable vogue as plague saints. In the successive epidemics of plague which ravaged Europe at distressingly frequent intervals throughout the Middle Ages and after, no explanation for the visitations was conceivable other than the wrath of a vengeful God. No stay of pestilence was known except by appeasing and propitiating the Deity. Processions, prayers, penitences, votive offerings, even sacrifices, were the only measures available against the appalling horror and misery. The intercession of the local saintly protectors was sought, as was also that of certain saints who possessed particular power against the plague. In the familiar painting by Titian, four of the most popular of the plague saints are portrayed. This picture was painted as a votive offering in gratitude for the recession of the plague of 1512 in Venice, and hangs in the sacristy of the Church of Santa Maria della Salute, itself a votive structure. In this painting, the central figure on the pedestal is Saint Mark, patron saint of Venice, still partly covered by a passing cloud, the shadow of the plague. Before him are Saints Sebastian, Roch, Cosmas and Damian. Sebastian, on the right, was the most powerful of the plague intermediaries. Because he had been sentenced to die by the arrow, he is always shown either holding arrows or pierced by them. Since remote antiquity, the arrow had been symbolical of the plague, and consequently, Sebastian had become associated in the popular mind with the plague, hence his influence against it. Next to Sebastian stands St. Roch, who won canonization because of a lifetime devoted to nursing plague sufferers. During these activities he developed plague and went into the woods to die. He was attended by an angel, however, and fed by his little dog, which went back into the city each day and returned with a loaf of

bread in its mouth, until he recovered. In paintings, Roch is always shown, as in this one, pointing to a bubo or an abscess in his

Fig. 5. Banner of the Confraternity of Saints Cosmas and Damian. (From Nicaise, Pierre Franco.)

groin. To the left in the picture stand the physician martyrs, Cosmas and Damian, also effective against the plague because of their association with the healing arts.

The most important role of Cosmas and Damian, and the one which interests us particularly, is that of patrons of medicine and surgery. Medicine in the Middle Ages, and long afterward, was entirely in the hands of the clerics, while surgery was a handicraft whose practitioners became incorporated into guilds. These organizations, like other guilds and companies, were placed under the protection of their tutelary saints. For this reason, the influence of the patron saints was more strongly felt in the field of surgery than in medicine.

It will be remembered that the clerical physicians were prohibited by the Council of Tours, in 1163, from practicing surgery by the famous dictum "ecclesia abhorret a sanguine." Surgery was therefore forced into lay and unlettered hands. The work of the barber most closely resembled that of

Fig. 6. First Coat of Arms granted to London Company of Barber-Surgeons in 1492. (From Young, Annals of Barber-Surgeons of London.)

the surgeon and the barbers assumed surgical practice. Throughout Europe guilds of barber-surgeons were organized, always under the patronage of Saints Cosmas and Damian.

A group of lay surgeons in Paris split very early from the barber-surgeon guild and formed the Confraternity of Saints Cosmas and Damian, later known as the Collège de St. Côme. This organization, said to have been founded in 1226 by Jean Pitard during the reign of St. Louis, was originally formed to limit by license those who were equipped to practice surgery. Later, it undertook to provide and supervise the education of surgeons, and in fact, became the Collège de St. Côme. The group adopted for its banner the figures of Saints Cosmas and Damian, shown with three gallipots. In the notice concerning the organization, (Fig. 5), which bears the date 1618, the privileges and obligations of the members are set forth. Of special inter-

est are the requirements that free service to the poor in need of surgical care should be given every Monday morning at the Collège, thus constituting a definitely established free surgical clinic.

SIGILLVM FACVLTATIS MEDICE VNIVERSITATIS PRAGENSIS
SIGILL FACVLTATIS MEDICÆ VNIVERSITATIS OENIPONTANÆ
SIGILLVM FACVLTATIS MEDICÆ
OMNIS MEDELA A DEO
SIGILLVM FACVLTATIS MEDICÆ
SIGILLVM MAIVS FACVLTATIS MEDICÆ

a *b* *c* *d* *e*

Fig. 7. Seals of the Medical Faculties of European Universities, showing Cosmas and Damian. *a*. Prague (1348). *b*. Oenipontana (1673). *c*. Wittenberg (1502). *d*. Leipzig (1409). *e*. Ingolstadt (1410).

Under the banner of the twin saints, the Collège de St. Côme fought a long, and eventually successful battle for the recognition of the dignity and independence of the surgical profession. The Faculty of Medicine disdained the menial tasks which surgical practice entailed. On the other hand, they were jealous of the powers and afraid of the ambitions of the surgeons, and desired a subservient and docile class of lay practitioners to follow their instructions in blood-letting, wound dressing and even in performing surgical operations. The barbers, who were organized into a guild of their own, sought to usurp the practice and prerogatives of the surgeons, and in this they were aided and abetted by the Faculty of Medicine. The medical group not only attempted to boycott the surgeons in favor of the barbers, but also tried to deny them the privilege of attending lectures, at the same time trying to provide lectures and demonstrations for the barbers. A three cornered controversy was thus waged between the surgeons of St. Côme on the one hand, and the Faculty and barbers on the other, which lasted almost five hundred years. In spite of repeated rebuffs and setbacks, the Collège persevered, and succeeded in wresting one concession after another, until, in the eighteenth century, the surgeons finally stood on a footing of equality with the physicians. The Collège de St. Côme became the Academy of Surgery, the forerunner of the present National Society.

A similar, but less stormy development took place in London. The Barber-Surgeons Company of London was founded in 1308 as a city guild. The first coat of arms granted to this Company in 1492 (Fig. 6) bears the images of Saints Cosmas and Damian on either side of the shield. The barbers and surgeons remained corporate bedfellows until 1745, when they were separated by an Act of Parliament. The Surgeons Company continued, and eventually, at the beginning of the last century, became the Royal College of Surgeons.

In the other countries of Europe, the guilds of barber-surgeons remained under the patronage of Cosmas and Damian. In the Netherlands, where master craftsmen suffered no social inferiority, the barber-surgeons were honored as much as any

other substantial bourgeois citizens. The pride and affluence of the chiefs of the Dutch barber-surgeons guilds of the sixteenth and seventeenth centuries are reflected in the many "Anatomy" paintings with which they adorned their guild halls. The most famous of these is the "Anatomy of Dr. Tulp" by Rembrandt.

In the German speaking countries, the scholarly education of surgeons developed very slowly, and until late in the eighteenth century surgery was practiced by the barber, the hangman, the sheep-shearer and the sow-gelder, as well as by itinerant operators and charlatans, all of whom paraded under the banner of Cosmas and Damian. Not until the universities undertook the teaching of surgery did the barbers surrender the practice, and only as surgery became a learned profession did the influence of Cosmas and Damian wane and give way to that of the disciples of modern science.

Even the early universities frequently chose Cosmas and Damian as medical patrons. Many of them, particularly in Central Europe, employed the Saints on the seals of their medical faculties. In Figure 7 may be seen several such seals, including that of the University of Prague, the oldest of the Central European universities, founded in 1348. Organized physicians also honored the martyred saints; the Medical Society of Vienna, as late as 1700, observed the Saints' Day with services in Stephan's Kirche, at which time pictures of Cosmas and Damian healing the sick were distributed to the populace.

Thus, whether we acknowledge as our craft ancestors the French Collège, the English Barber-Surgeons' Company or the guilds of barbers and surgeons of Germany; and whether we trace our evolution from the crafts of shearers and field surgeons, or from the faculties and universities, we find Saints Cosmas and Damian the patron saints of our forbears, and can trace their patronage from remote beginnings in the early Dark Ages almost down to our own irreverent generation.

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BOOK REVIEWS

AN INDEX OF DIFFERENTIAL DIAGNOSIS OF MAIN SYMPTOMS. By various writers. Edited by Herbert French, C.V.O., C.B.E., M.A., M.D., F.R.C.P. Fifth Edition with 742 illustrations of which 196 are coloured. Baltimore, William Wood and Company, 1936.

This monumental work needs no introduction to the medical profession of this country as for over two and a half decades it has enjoyed a large sale both here and abroad. From those who have had this book on their library shelves we learn that it is a frequent source of reference and a very satisfactory one, at that.

It is a book that almost defies a proper review. It is impossible to give an idea of the scope of the book or linger on any of its details. We meant to spend a half-hour getting a general idea of the work, and then spent a whole evening, and far into the night, looking up this and that subject. Written by nineteen authors, each one of national repute, the work is distinguished by good writing plus an adequate consideration of the subject to be treated. Anyone practicing medicine will find it invaluable.

Although a large book, it has been printed in readable type on good paper, and the illustrations are above the average. The colored plates are exceptionally good. The index, alone, is a book in itself.

This is one book we cannot help but urge the other fellow to possess provided he wants or needs an exceptional work on differential diagnosis.

POST-GRADUATE SURGERY. Edited by Rodney Maingot, F.R.S.C. (Eng.). With an introduction by the Right Hon. Lord Moynihan of Leeds. Vol. I, with 846 figures and Vol. II with 1134 figures in the text. New York, D. Appleton-Century Company, 1936.

This is a new work that when completed will comprise three volumes. We await the third volume with keen anticipation.

Dr. Eugene H. Pool did the "American Foreword." Among other things Dr. Pool says that "One must feel encouraged by evidence that times have improved as shown by this presentation to the profession of a great comprehensive surgery. The surgeon must also feel an urge to replenish his library with something new and stimulating."

This work is not a textbook but is intended for the postgraduate student and practicing surgeon. It covers a wide range, practically the whole field of surgery. Naturally, it includes technique but emphysis is stressed in preoperative and postoperative treatments.

The first volume of 1741 pages, is devoted to: Part 1. Anesthesia, General, Local, and Choice of Anesthesia; Part 2. Abdomen—there are 14 sections, by various writers; Part 3. Rectum and Anus; Part 4. *X*-Ray Diagnosis; Part 5. Radium Treatment. Volume II has 1831 pages and is divided into eleven parts: Head, Spinal Column, and Salivary Glands; Neck; Breast; Female Genital Organs; Urinary System and Male Genital Organs; Sympathetic Nervous System; Adrenal Gland; Injection Therapy; Infections of the Hand; Orthopedics. There is an Index.

Inasmuch as the text is from the pen of men well known in their country but not so well known to many in the United States, their viewpoint is refreshing, even though in minor details many of our collegues will find fault and differ. But we agree with Dr. Pool and found the work "something new and stimulating." We hope it "catches on" in this country as it deserves, for it is in a class by itself and decidedly important and valuable.

ABORTION. SPONTANEOUS AND INDUCED. MEDICAL AND SURGICAL ASPECTS. By Frederick J. Taussig, M.D., F.A.C.S. Illustrated. St. Louis, C. V. Mosby Company, 1936.

This volume, which is one of a series dealing with medical aspects of human fertility, was sponsored by The National Committee on Maternal Health, Inc. Do not let this statement scare or prevent you from reading this book. It is a timely topic for the subject of abortions enters the daily practice of most physicians.

Dr. Taussig has done a valuable work. We did not merely glance through it, but practically read every line. To our mind it is the last word on the subject and nothing more need be written until that time when something new on the subject has been discovered or developed.

Surely every gycologist and obstetrician will do well to study the text, and for the fellow in the smaller places whose work takes him into many fields, if he does work with pregnant women, then the book becomes almost an essential for him.

Every phase of abortion is considered and treated in detail. The presentation is orderly, clearly written, and there is an Appendix, *a*. Statutes Relating to Abortion; *b*. Source Tables; *c*. Glossary of Terms, an Index and a satisfactory Bibliography.

A needed work, well done.

AN INTRODUCTION TO SURGERY. By Rutherford Morison, Emeritus Professor of Surgery, Durham University, and Charles F. M. Saint, Professor of Surgery, Cape Town University, South Africa. Third Edition. Baltimore, William Wood and Company, 1935.

This book should be valuable to senior medical students, internes and young men learning the art and science of surgery. As we read in the Introduction to the First Edition, "No one would think of building a house without first providing a firm foundation but this, in effect, is what the student is apt to do if his methods of learning consists in attempting to memorize the mass of detail to be found in the average text-book." This book will aid the student in solving the problems presented to him.

Inasmuch as few students will see this review, it might not be presumptuous for us to suggest that those who teach surgery to undergraduates might look this book over, as a new idea and viewpoint are always refreshing, and if it will not add too great a burden, it might be recommended to their classes.

MEDICAL PAPERS. Dedicated to Henry Asbury Christian, Physician and Teacher. From his present and past associates and house officers at the Peter Bent Brigham Hospital, Boston, Mass. In honor of his Sixtieth birthday, February 17, 1936. Baltimore, Waverly Press, Inc., 1936.

When an individual, during the course of a successful and full life, reaches the "middle years," for such are those of a man of sixty, it thrills anyone in medicine when that man's associates and former pupils honor him. It is the custom to place bouquets, often accompanied by maudlin sentiments, to the memory of the departed, but it seems, more fitting, especially to the reviewer, to honor one who has reached the heights while among the living.

Doctor Christian must feel deeply the appreciation and esteem in which he is held by those who have worked during many years with and under him. And this volume of 1000 pages is a fitting and worthy expression of that devotion and admiration.

Usually volumes of this type are composed of articles, for the most part, which just miss being worthwhile. In this sense this volume is the exception to the rule. There are 103 original articles and an introduction, but, we cannot go into detail and discuss any article. Many of the writers are men of wide reputation, and each article bears the stamp of careful, thorough preparation and work.

This volume was not published for sale and gain. Only a few copies are available by purchase. We can say without hesitation that merely as a book composed of various articles of a miscellaneous character it would be worth the cost. Any physician will find much between its covers to his benefit.

This expression of esteem and affection, to one who has meant so much to medicine in this country and to the Harvard University Medical School, is one of the fine things that happen too seldom in life.

A TEXTBOOK OF SURGERY. By American authors. Edited by Frederick Christopher, B.S., M.D., F.A.C.S. With 1349 illustrations on 730 figures. Philadelphia, W. B. Saunders Company, 1936.

Nearly everybody who is in, or should be in, "Who's Who" in American surgery has contributed to this book. As a rule, books of this type are in the main unsatisfactory and a medley of disassociated articles. For once we have an exception to the rule, and it is a joy to the reviewer to tell you that this is an exceptional work.

Also, it lives up to the claims made for it by Dr. Christopher in the Preface. He says, "The dominant plan of this textbook is to give the student a concise presentation of surgery which is characterized by the maximum authority. . . . The subject matter contains the tested and accepted present day principles of surgery." Anyone who carefully reads this work will agree with these statements.

Therefore, this being true, it is apparent that this well indexed book of 1608 pages, is ideal for the purposes of the student and the practicing surgeon. It warrants and, no doubt, will have an enthusiastic reception by the profession.